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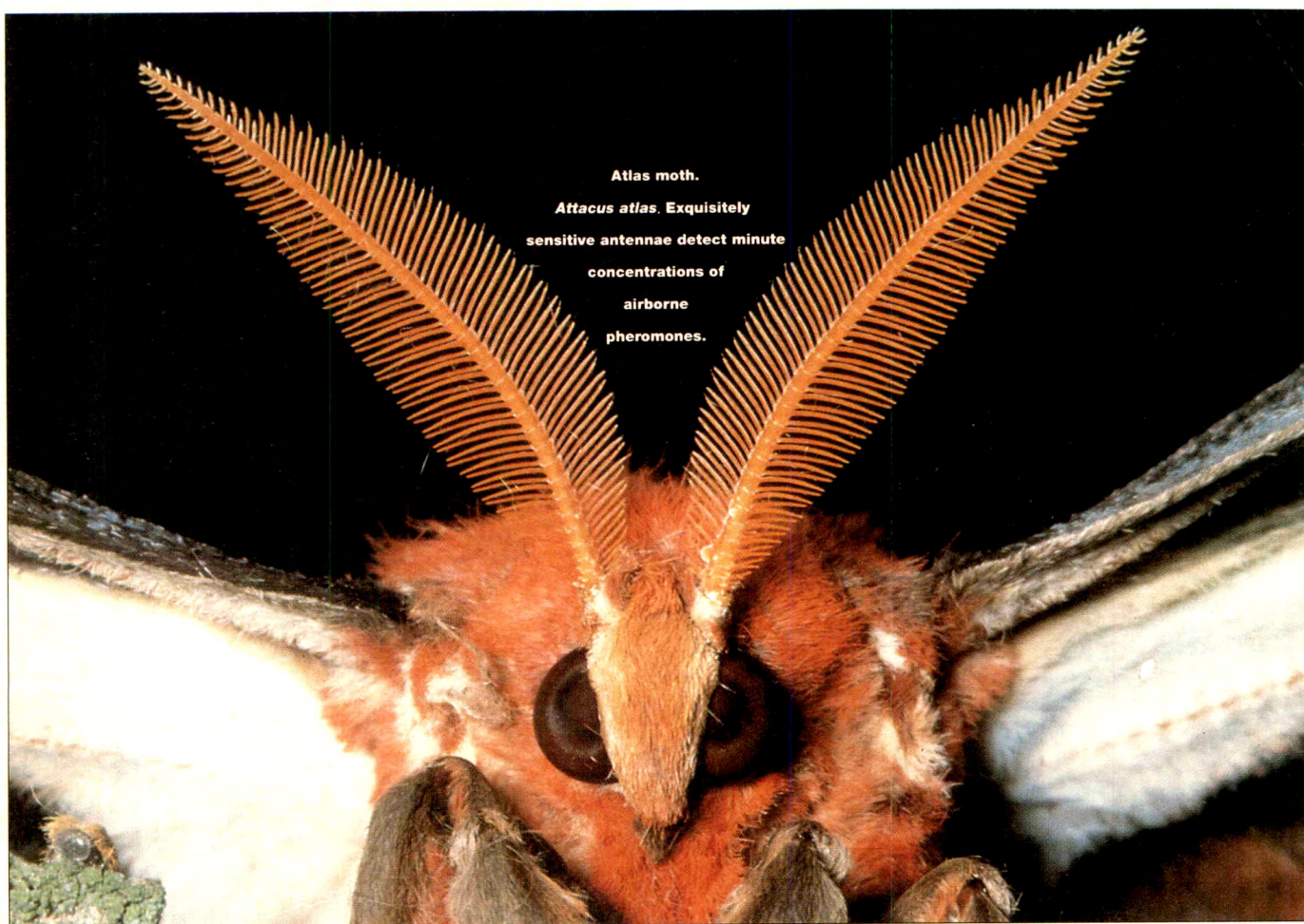
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Atlas moth.
Attacus atlas. Exquisitely
sensitive antennae detect minute
concentrations of
airborne
pheromones.

Is this an anaesthetist's role model?

P 24,533

*An anaesthetist, like a moth,
develops a sensitivity bordering on "sixth sense";
mental antennae attuned to every
requirement of the patient.*

Diprivan▼
propofol

i.v. anaesthesia

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▼Special reporting to the CSM required.

Consult data sheet before prescribing.

Use: Induction and maintenance of general anaesthesia for surgical procedures lasting up to one hour.

Presentation: Ready to use, isotonic, aqueous emulsion containing 10mg/ml propofol in a vehicle containing soybean oil and purified egg phosphatide.

Dosage and administration: **Induction:** Titrate against response using approximately 4ml every 10 seconds in healthy adults and 2ml every 10 seconds in patients of ASA grades 3 and 4. Patients under 55 years are likely to require 2.0 to 2.5mg/kg, older patients may require approximately 20% less.

Maintenance: Usually 0.1 to 0.2mg/kg/min (6 to 12mg/kg/hr). Continuous infusion may require slightly higher rates for 10 to 20 minutes after induction. The infusion can be used undiluted or diluted with 5% Dextrose (intravenous infusion BP). Alternatively repeat bolus injections of 25 to 50mg (2.5 to 5.0ml) may be administered according to clinical need.



Paediatric Use: No experience in children or in mothers who are breast feeding.

Contraindications: Allergy to 'Diprivan'.

Precautions: Ampoules and vials should be inspected before use for particulate matter and discolouration. Aseptic technique should be followed throughout its handling; administration commencing without delay once drawn into the syringe or giving set. Discard any unused 'Diprivan' after single patient use. Do not mix prior to administration with other agents or infusion fluids other than Dextrose 5%, such dilutions should be prepared immediately before administration. Hypotension and transient apnoea may occur during induction. Occasionally, hypotension may require use of i.v. fluids and a lower rate of administration during maintenance. Apply caution in cardiac, respiratory, renal or hepatic impairment; epilepsy; in hypovolaemic or debilitated patients; and in disorders of fat metabolism or conditions where lipid emulsions should be used cautiously. Bradycardia may occur due to the lack of vagolytic activity of 'Diprivan' and administration of an anticholinergic should be considered particularly if vagal

tone is dominant. Do not use in pregnancy except for termination. Discharge after general anaesthesia - allow adequate time for full recovery.

Side effects: Epileptiform movements, including convulsions and opisthotonus, have occurred rarely in a temporal relationship to 'Diprivan'. Nausea, vomiting and headache in a small proportion of patients. Very rarely, clinical features of anaphylaxis, which may include bronchospasm and erythema accompanied by hypotension, have been reported with 'Diprivan'. Pain on injection in a proportion of patients. Discolouration of urine, venous sequelae and fever are rare. Minimal evidence of excitation on induction.

Product licence number: (29/0190). **Basic NHS cost:** £3.98 per 20ml ampoule, £9.95 per 50ml infusion vial.

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Anaesthesia

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Editorial

The future development of chronic pain relief

There is now a pain clinic in almost every general hospital in the United Kingdom, and the number in the Republic of Ireland is increasing. Almost all these clinics are either run by an anaesthetist or have a consultant anaesthetist among their staff. However, we are training nowhere near enough people to fill these posts. There is no nationally recognised training programme; indeed in many regions there is no training programme at all. The Department of Health will recognise that a new subspecialty has come into existence and will collect data about its activities, but will only fund them when it has developed a formalised training programme. Thus although the clinics already exist, their activities are not separately recorded and so development can only be haphazard and the future insecure.

The demand for the services of pain clinics is overwhelming; a recent College of Health household survey found that 11% of those surveyed had unrelieved pain of more than 3 months' duration. Some of these patients may have a disease process whose treatment will result in relief of their pain. However, most will not and they will be suffering from such things as chronic back pain, headache, facial pain, postherpetic neuralgia, central poststroke pain, fibromyalgia and pain resulting from incurable disease. The only methods that will have any prospect of giving these patients relief are those aimed at the symptomatic alleviation of their pain.

The ability to analyse chronic pain, relate it to pain physiology, to assess and apply the known methods of chronic pain relief and management, make up a separate and new branch of medicine, pain relief. The majority of those who have formed and developed this new branch of medicine are anaesthetists. If the College of Health survey represents a true national figure 5 000 000 people, (25 000 in a medium sized health district) might benefit from their services. It is surely a basic tenet of our profession that the relief of pain and suffering should be paramount. Why is there no training programme so that the Department of Health can proceed and develop this service? The answer cannot be that the topic does not matter. Is it because there is a suspicion that running a pain clinic is rather pointless and does little good, an occupation for the dilettante who would be more usefully employed in theatre being a 'proper' anaesthetist? The knowledge of pain physiology has increased dramatically in the 25 years since pain clinics were first established and with it, but more slowly, a rationale for treatment and an improvement in results.

Historically, anaesthetists involved in the management of chronic pain offered their skills in the management of terminal cancer by nerve blockade, as an alternative to the use of large doses of morphine which was regarded as dangerous and wrong. Once established as pain relievers anaesthetists found themselves confronted with a large and demanding case load and used a number of regional techniques whose usefulness had not been validated in properly controlled trials in the management of chronic pain. Essentially applied physiologists and pharmacologists, these

anaesthetists found that a majority of the conditions they were treating had an undefined pathology and that treatment methods and results depended largely on anecdotal recommendation; an understanding of the physiology was a matter of debate rather than proven fact. This was no basis for the development of an advancing branch of medicine and perhaps many believe that this is where pain relief has stuck. This belief is no longer tenable and may be interfering with the development of services and training in this area.

The knowledge of pain physiology and its application to the relief of pain is increasing continually. The starting point of much of modern pain management was the publication of the gate theory.¹ This suggested that pain is perceived partly as a result of the nature of the information reaching the central nervous system from specific receptors and partly as a result of modulation generated within the central nervous system (CNS) through other incoming sensory information and descending inhibition. The possibility that pain might result either from stimulation of pain receptors (nociceptors) or that pain might be perceived when the CNS pain perception mechanisms were damaged developed from this concept. Thus pain may be felt either as a result of tissue damage (nocigenic) or as a result of a defective modulation of incoming sensory information by the peripheral, autonomic or central nervous systems (neurogenic).² The presence of pain in the absence of detectable tissue damage is therefore just as real as any other and it is frequently beyond our current ability to analyse its source within the central nervous system.

The modern pain clinic approach to the relief of pain has developed from this base using either blockade of afferent nocigenic stimulation by local anaesthetics or other analgesics, or through attempting to alter neuronal function, to reduce neurogenic pain by the use of nonanalgesic drugs such as anticonvulsants and antidepressants. Where pain is the result of tissue damage the techniques of acute and chronic pain relief do not differ and it is their efficient application rather than the techniques themselves that is new. The situation is not yet quite so straightforward, however; intractable back pain, which imposes a heavy load on the community and on pain clinics is apparently nocigenic, but we remain almost entirely ignorant of the site of the tissue damage so its management remains empirical. This is not a unique situation in modern medicine and the simple local anaesthetic methods of back pain treatment such as epidural injection³ have been validated as beneficial and do no harm;⁴ back surgery is no more effective and on occasion may be accompanied by permanent complications. Techniques such as transcutaneous electrical nerve stimulation (TENS) and acupuncture are used in the management of both nocigenic and neurogenic pain, although their evaluation remains extremely difficult because of the power of the placebo effect, their nonspecific nature and the difficulties involved in constructing controlled

trials.^{5,6} They play an important part in the management of the previously intractable neurogenic pain syndromes, such as postherpetic neuralgia, central poststroke pain and pain resulting from damage to the spinal cord, which are becoming more clearly understood as their characteristics are more precisely defined.⁷ In several of these syndromes antidepressant drugs are of benefit, although the role of the anticonvulsants remains debatable.⁸ The interaction of the sympathetic system and the sensory nervous system in the generation of sympathetically dependent pain (causalgias) is also slowly becoming clearer⁹ and with it a rationale for the use of sympathetic blockade, a technique that may also have a place in fibromyalgia.¹⁰ When the symptom of pain remains despite medical intervention patients can be returned to the community as intact social and working people through the rehabilitative techniques of pain management.

The development of pain management has resulted from our understanding of the influence that suffering unrelieved pain has on behaviour. Patients complaining of pain for which we can find no organic cause are psychologically similar to patients with chronic pain for which an organic cause exists; however, the presence of unrelieved pain causes changes in behaviour that reinforce the resulting invalidity.¹¹ Pain management programmes use behavioural methods to reduce this altered behaviour and with it the invalidity resulting from chronic pain.¹²

Thus in the main areas of pain clinic activity a knowledge of pain physiology, neuropharmacology and psychology is being applied increasingly systematically to improve the management of this exceptionally demanding problem. It is no longer largely empirical, anecdotal and a waste of time. The work is being done almost entirely by anaesthetists and has been for the last 25 years. It must be time for the specialty to formalise a training programme. This would be followed by a

recognition of the existence of pain clinic activity by the Department of Health, the training of a more appropriate number of potential pain clinicians and finally some relief for those suffering from chronic unrelieved pain.

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A. DIAMOND

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Lumbar epidural diamorphine following thoracic surgery

A comparison of infusion and bolus administration

J. A. PATRICK, M. MEYER-WITTING AND F. REYNOLDS

Summary

Twenty-two patients received a single dose of diamorphine 5 mg through a lumbar epidural catheter before thoracic surgery. The patients were transferred after surgery to a high dependency unit where they were allocated randomly to receive either an infusion of epidural diamorphine at a rate of 1 mg/hour (group 1) or bolus doses of epidural diamorphine 5 mg on demand (group 2). There was no statistically significant difference between the groups in visual analogue pain scores in the first 18 postoperative hours. Arterial carbon dioxide tension was elevated in both groups and was consistently higher in group 1 than in group 2, with a statistically significant intergroup difference 12 hours after operation. Respiratory rate was not a useful index of respiratory depression. The commonest nonrespiratory side effect was urinary retention, but the incidences of this and other minor side effects were similar in the two groups.

Key words

Pain; postoperative.

Analgesics; diamorphine.

Anaesthetic techniques, regional; epidural.

Thoracic surgery ranks high in the league table as a cause of severe postoperative pain,¹ and several approaches have been used in an attempt to improve analgesia and respiratory function.² Epidural administration of local anaesthetics may provide good analgesia,³ but side effects, especially hypotension and urinary retention, are common.³⁻⁵ In addition, the epidural catheter must be positioned in the midthoracic rather than the lumbar region in order to avoid high dose requirement and superfluous lumbosacral blockade. This approach is technically more difficult and even in experienced hands has relatively high complication⁴ and failure⁶ rates.

Epidural opioid administration has the advantages of selective spinal analgesia without measurable sympathetic or motor block, and has become well established in the treatment of postoperative pain.^{7,8} In addition, morphine can be given in the lumbar region to treat thoracic and upper abdominal pain.⁹⁻¹¹ At this hospital, diamorphine is preferred to morphine because its greater lipid solubility results in more rapid onset and less risk of unpredictable late respiratory depression.¹² Furthermore, it comes as a convenient preservative-free preparation, unlike morphine. The aim of this study was to compare the use of a continuous lumbar epidural infusion of diamorphine with intermit-

tent bolus doses in the treatment of pain after thoracic surgery.

Methods

The study was approved by the local ethics committee and each participant's consent was obtained. Twenty-three patients who underwent thoracic surgery were studied. An epidural catheter was inserted in the second lumbar interspace after induction of anaesthesia, and a bolus dose of diamorphine 5 mg dissolved in 10 ml of physiological saline was administered to all patients through the catheter. No other pre- or intra-operative opioids were given. Anaesthesia was maintained using nitrous oxide and enflurane in oxygen. Peripheral venous and arterial cannulae were inserted in all patients, and an internal jugular vein was cannulated in patients who underwent lobectomy or pneumonectomy.

The patients were transferred at the end of surgery to a postoperative high dependency unit (HDU) where heart rate, arterial blood pressure, respiratory rate and arterial blood gases were measured hourly without disturbing the patient. Patients were nursed in the semisitting position and breathed humidified oxygen-enriched air. The patients were

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Table 1. Patient details.

	Group 1	Group 2
<i>n</i>	11	11
Males	6	7
Smokers	7	6
Mean age; years	50.4	49.7
(range)	(20–67)	(29–65)
Mean weight; kg	64.0	73.0
(range)	(47–88)	(45–96)

allocated randomly to one of two groups. Those in group 1 received an epidural infusion of diamorphine 10 mg dissolved in 50 ml of normal saline, which was started on arrival in HDU and administered initially at a rate of 1 mg/hour; patients in group 2 received bolus doses of epidural diamorphine 5 mg on demand. An initial bolus dose of diamorphine 2.5 mg was given through the epidural catheter to any patient in group 1 who stated that analgesia was inadequate. The infusion was discontinued temporarily if the respiratory rate was less than 10 breaths/minute, or in the event of a progressive increase in arterial PCO_2 ; it was restarted at half the previous rate when these measurements became acceptable. Pain was assessed using an ungraded 10-cm visual analogue scale at 1, 2, 3, 4, 6, 8, 12, 15 and 18 hours after arrival in HDU. All patients were transferred to a general cardiothoracic surgical ward on the day after surgery.

The statistical significance of differences between groups was tested using Student's *t*-test, Mann-Whitney *U*-test and the Chi-squared test as appropriate.

Results

The intra-operative use of a single bolus dose of epidural diamorphine 5 mg provided satisfactory analgesia during surgery without the need for supplementation with other opioids.

One of the 23 patients recruited was withdrawn from the study because epidural diamorphine produced no analgesia and epidural bupivacaine 0.5% produced neither sympathetic nor sensory block. The distribution of patients to the

two groups was similar in respect of gender, age, weight and smoking history (Table 1). Eight patients in each group underwent thoracotomy with lobectomy or pneumonectomy and three had thoracoscopy and pleurodesis.

Visual analogue scores recorded by the patients are shown in Figure 1. There were no statistically significant differences between the groups at any point during the 18-hour study period ($p > 0.05$, independent *t*-tests). The mean (SD) total peri-operative dose of diamorphine was similar in the two groups: 21.9 (5.4) mg (range 13–28 mg) in group 1 and 20.0 (5.9) mg (range 10–30 mg) in group 2. Seven of the patients in the infusion group required one supplementary bolus dose of diamorphine 2.5 mg within 2 hours of arrival in HDU; another two patients required two supplements within the first 4 hours of the study period. The mean time between requests for the first and second postoperative doses of diamorphine was 6.7 (4.2) hours in 10 patients in group 2. The remaining patient required only one dose of diamorphine 5 mg during the 18-hour study period. No other analgesic was given to any patient in either group.

Mean respiratory rate at hourly intervals is shown in Figure 2. There was no statistically significant difference in respiratory rate between the groups at any time ($p > 0.05$ Mann-Whitney *U*-test). Six patients in group 1 and three in group 2 had one or more recorded respiratory rates of less than 10 breaths/minute during the first 18 postoperative hours. Mean $Paco_2$ values (Fig. 3) were higher at all times in group 1 than in group 2 during the 18-hour study period. The difference was significant at 12 hours (group 1, 6.76 (0.69) kPa; group 2, 5.85 (0.51) kPa; $p = 0.005$, Mann-Whitney *U*-test). $Paco_2$ fell progressively in both groups during the study period, but the mean value was consistently greater than 6 kPa in the infusion group. There was no useful correlation between respiratory rate and $Paco_2$ (Fig. 4).

All patients breathed supplementary oxygen when appropriate. Four patients in group 1 had a single recorded oxyhaemoglobin saturation value of less than 90%, with a minimum of 88.6%. Three were breathing room air at the time and the FIO_2 of the fourth was 0.28. The only recorded value less than 90% in group 2 was one reading of 84.5% ($PO_2 = 6.7$ kPa) with an FIO_2 of 0.4 in a 48-year-old female

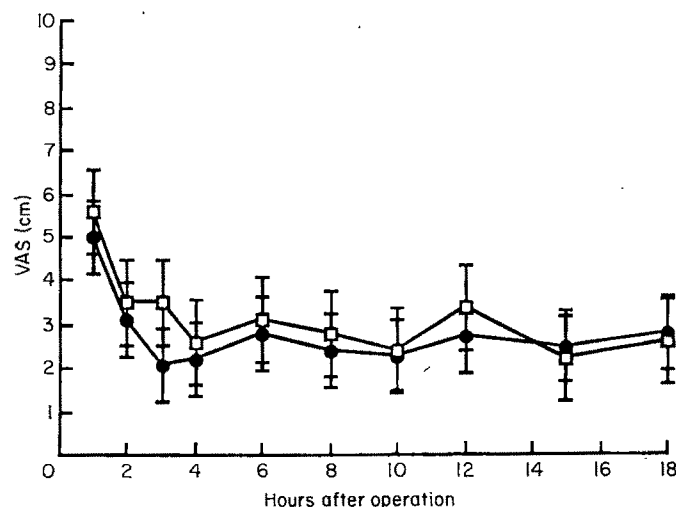


Fig. 1. Mean (SD) visual analogue scores (VAS) recorded by patients in group 1 (infusion; ●) and group 2 (bolus; □) during the first 18 postoperative hours.

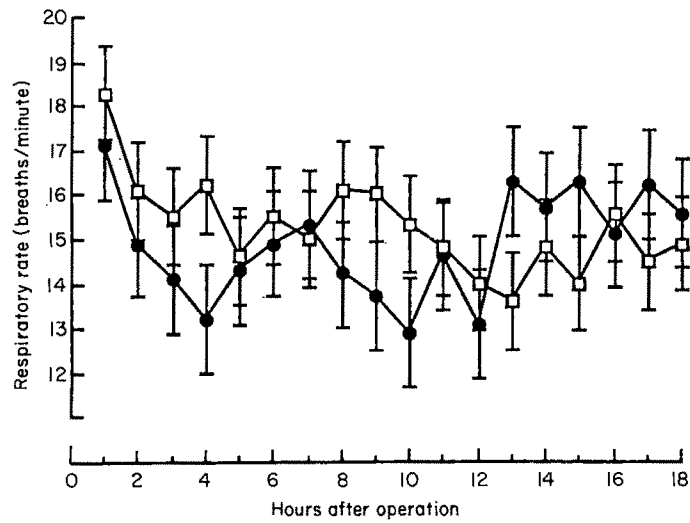


Fig. 2. Mean (SD) respiratory rate of patients in group 1 (infusion; ●) and group 2 (bolus; □) during the first 18 postoperative hours.

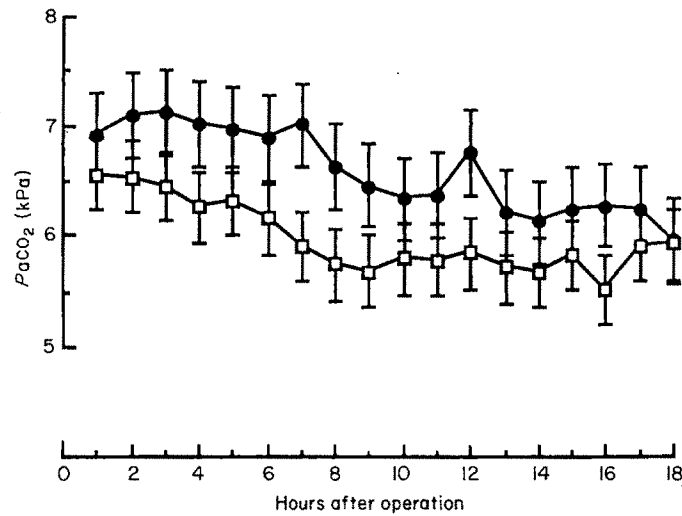


Fig. 3. Mean (SD) arterial carbon dioxide tension in group 1 (infusion; ●) and group 2 (bolus; □) groups during the first 18 postoperative hours.

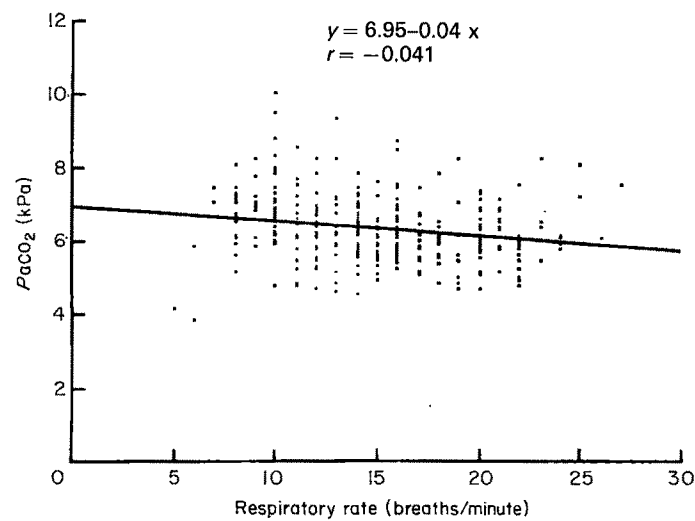


Fig. 4. Scatter plot relating arterial carbon dioxide tension to respiratory rate at the time of arterial blood gas measurement (results from both groups).

Table 2. Nonrespiratory adverse effects.

	Group 1	Group 2
Drowsiness	11	11
Urinary retention/bladder catheterisation	9	8
Itching	2	2
Vomiting	1	0
Nausea	1	3
Stroke	0	1
Paralysis	1	0
Persistent hypotension	1	0

with progressive pulmonary fibrosis; $Paco_2$ at that time was 5.6 kPa and her respiratory rate was 16 breaths/minute.

Two patients suffered serious neurological events. A 61-year-old woman in group 1 who had a lobectomy was found to be paraplegic 2 days postoperatively (although the onset may have been earlier), with a level of T_6 , the level of her thoracotomy. This is a rare complication of thoracotomy,¹³ and we do not believe that it was related to lumbar epidural diamorphine administration. A 63-year-old man in group 2 who had a pneumonectomy was given bolus doses of diamorphine 5 mg 30 minutes and 9 hours after operation. His vital signs were stable and satisfactory and his arterial blood gases were good. He suffered a stroke while being turned by the nurses 3 hours after the second dose. This was thought to have been a sudden embolic episode. He was given naloxone empirically, but did not respond to it.

Only one other patient was given naloxone during the study period. A 61-year-old man in group 1 became difficult to rouse 4 hours after surgery. His $Paco_2$ was 9.3 kPa, pH 7.23, PaO_2 29.1 kPa ($F_{IO_2} = 0.5$) and respiratory rate 13 breaths/minute. He was given naloxone 0.1 mg and his $Paco_2$ decreased to 7.0 kPa one hour later when he was more awake.

Nonrespiratory adverse effects are shown in Table 2. Urinary retention was the commonest side effect, and occurred in 77.3% of patients, affecting males and females equally. The incidences of this and other minor side effects such as pruritus, and nausea and vomiting, were similar in the two groups and episodic drowsiness was a universal finding.

Discussion

The administration of lumbar epidural diamorphine by infusion did not produce better analgesia than bolus doses in our patients during the first 18 postoperative hours. The advantage of infusion analgesic techniques should be more consistent analgesia, but supplementation with epidural bolus doses^{9,14} or systemic opioids^{5,15} may be required in patients who receive an epidural opioid infusion, even when loading doses have already been given. Seven patients in our infusion group required supplementary doses. Epidural infusions may be more appropriate if shorter acting agents such as fentanyl are used,^{16,17} because although they act rapidly, they also wear off relatively quickly.

There was no difference in respiratory rate between the groups during the study period but mean arterial carbon dioxide tension was higher in the infusion group. The $Paco_2$ values are higher than those reported by Cooper *et*

*al.*¹² after postoperative epidural administration of diamorphine. However, their study was conducted on the first postoperative day, not on the day of surgery, and in patients who had had abdominal rather than thoracic surgery. In another study,¹⁸ end-tidal CO_2 values measured during thoracic epidural infusion of fentanyl in the first 18 hours after a variety of procedures appeared to be low. However, measurements of end-tidal CO_2 concentration using an intranasal cannula may be deceptively low¹⁹ especially when, as in this case, the cannula was inserted only 2 cm into the nares.

Our values for $Paco_2$ are comparable to those found by Asantila *et al.*²⁰ who used a variety of forms of analgesia, including epidural morphine, on the day of thoracotomy, a surgical procedure known to cause marked changes in pulmonary function.²¹ In addition, the physiological changes which occur during sleep²² may make some contribution to alterations in measured blood gases on the night of surgery.

It is now well established that in common with opioids given by other routes, spinal opioids cause respiratory depression.²² However, respiratory rate is an unreliable index of respiratory depression, because the relationship between measured arterial blood gases and respiratory rate is not consistent.²³ This has been confirmed in our study, where some patients with marked hypercapnia had acceptable respiratory rates (Fig. 4); the only patient thought clinically to require naloxone for respiratory acidosis had a respiratory rate of 13 breaths/minute, but was unresponsive to all but painful stimuli. Clearly, a low respiratory rate requires action, but a normal respiratory rate should not be taken to mean that all is well. The patient's conscious level should also be assessed. Significantly altered consciousness, in contrast to the more common drowsiness, is an important concomitant of respiratory depression in postoperative patients who receive epidural opioids²³ and may be detected more readily in HDU. Arterial blood gas analysis can then be performed when appropriate.

The most commonly reported nonrespiratory side effect in the first 24 hours after surgery, apart from episodic drowsiness, was urinary retention requiring catheterisation, a well documented feature of epidural opioid administration.

The incidence of pruritus (18%) in both groups was low compared to previous studies of epidural diamorphine following Caesarean section. Macrae *et al.*²⁴ and Semple *et al.*²⁵ recorded an incidence of itch of 36% and 50% respectively. However, Jacobson *et al.*²⁶ reported no pruritus with the same drug and route of administration after thoracic and lower abdominal surgery. Patients may be less aware of relatively minor nonpainful symptoms such as itching during recovery from general anaesthesia. It has been stated that the pruritus that occurs after epidural diamorphine is often not severe enough to require treatment and may be elicited only on direct questioning.²⁵ None of the patients in our current study required treatment for pruritus.

In conclusion, an infusion offered no clinical advantage when lumbar epidural diamorphine was used following thoracic surgery. Arterial carbon dioxide tension was elevated in both groups, more so with infusions, but by the end of the 18-hour study period, $Paco_2$ was the same in both groups and was returning towards normal values. Thus respiratory impairment was present but the fact that

it ameliorated during the period of study suggests that surgery and anaesthesia were contributory factors. Respiratory rate alone was not a useful index of respiratory depression. The ideal of continuous noninvasive monitoring which detects hypoxaemia, apnoeic episodes and severe hypoventilation has not yet been realised,²² but the importance of a decreased level of consciousness has been stressed.²³ At present, the safest method of monitoring patients who receive epidural opioids after major surgery appears to be close nursing supervision in a high dependency unit. However, it should be remembered, especially in view of the current close scrutiny of spinal opioids, that administration of the same drugs by other, perhaps less effective routes, is not without hazard.²⁷

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Intra-operative patient-controlled sedation

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Summary

Patient-controlled sedation using propofol is described in 23 ASA I patients undergoing surgical extraction of third molars. Cardiovascular and respiratory stability was maintained and sedation was no deeper than full eyelid closure with prompt response to verbal command. Amnesia for the extractions was common (70%), but there was no significant amnesia for recovery room events. Intra-operative patient cooperation was good, postoperative patient satisfaction high and all patients stated they liked the self-administration and would use the same technique again. Propofol dose was correlated with both procedure length ($r = 0.587$; $p = 0.003$) and surgical difficulty ($r_s = 0.550$; $p = 0.010$).

Key words

Anaesthetics intravenous; propofol.
Surgery; dental.

Intra-operative sedation to augment local or regional anaesthesia should include optimal patient satisfaction with minimal risk of morbidity and mortality.¹ This can be difficult to provide because of pharmacokinetic and pharmacodynamic variability,² different interpatient requirements, poorly defined patient expectations and changing intra-operative conditions.

Patient-controlled analgesia has been used with success to overcome similar problems in postoperative analgesic requirements.³ We have undertaken this study to determine if such techniques offer advantages in the provision of safe and effective intra-operative sedation. The pharmacokinetic properties of propofol,⁴ particularly its rapid onset, redistribution and metabolism and its low incidence of side effects in the lipid formulation⁴ make it a suitable agent to consider for patient-controlled sedation (PCS).

Satisfactory intra-operative sedation should also include suitable postoperative recovery characteristics. Delay in recovery of mental function and memory may, in particular, be disadvantageous for day surgery patients. Sedation with propofol by infusion has been associated with faster recovery and less postoperative amnesia than sedation with intermittent diazepam⁵ or midazolam by infusion,⁶ supporting its use as the preferred agent for intra-operative PCS.

We have used the surgical extraction of lower third

molars (usually in association with the additional extraction of upper third molars) to provide a reproducible range of stimulation in patients who are apprehensive. The procedure is often stressful in spite of effective local anaesthesia, and provides a strong indication for sedation. On the other hand, sedation should not be excessive, to avoid airway difficulties with secretions and blood in the presence of depressed reflexes, whilst still maintaining patient cooperation.

Methods

The study was approved by the hospital Human Ethics Committee and all patients gave informed consent. All operative procedures were carried out by one surgeon (N.C.).

Twenty-three unpremedicated, fasted, day surgery patients received patient-controlled intravenous propofol using a commercially available syringe pump (Graseby Medical Ltd) modified by the manufacturers to provide a minimum lockout period of one minute. An initial dose of 0.7 mg/kg of propofol was administered over 15 seconds by the anaesthetist before the administration of dental local anaesthesia. This dose was chosen following pilot studies in patients of similar age, weight and ASA classification undergoing third molar extractions. Thereafter, by means

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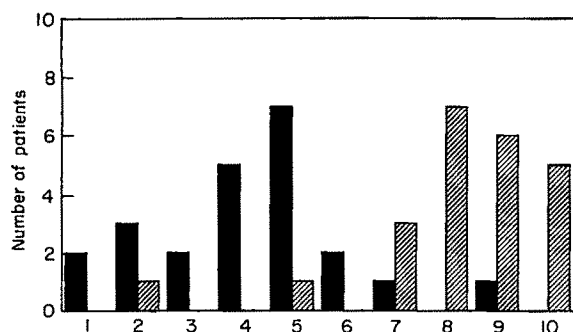


Fig. 1. Distribution of scores for surgical difficulty (■) and patient cooperation (▨).

of a hand-held triggering device, patients self-administered bolus doses of 0.7 mg/kg delivered at a rate of 16.7 mg/minute. Patients were informed that pressing the button would deliver medication that would make them sleepy, and that if they wished to be more sleepy at any time then they should further press the button. The lockout period to guard against overdose was also explained and they were told that an anaesthetist would be present at all times. Patients were excluded from the technique if the expected degree of surgical difficulty was excessive for local anaesthesia and sedation (judged from radiographs), if they suffered from allergy, or had significant psychiatric illness, difficulty with communication or an ASA status greater than 2.

Each procedure was scored 0–10 by the surgeon for surgical difficulty (maximum 10) and patient cooperation (maximum 10).

A 22-gauge cannula inserted into a peripheral vein was used for drug administration and intravenous fluids. All patients received supplementary oxygen by nasal cannulae and 1 litre of 4% dextrose with 0.2 N saline peri-operatively. The infusion sets contained a one-way valve to

Table 1. Summary data for patients and procedures. Values expressed as mean (SD).

Age; years	21.7 (3.8)
Weight; kg	58.6 (10.7)
Procedure time; minutes	44.3 (13.2)
Recovery time to discharge; minutes	122.0 (35.3)

prevent reflux of propofol. The ECG, noninvasive arterial blood pressure and pulse oximetry were monitored by a multifunction monitor (Datex Cardiocap Model COS14) and intra-operative heart rate, arterial pressure and haemoglobin saturation were recorded on a videographic printer (Sony Model UP-701).

Times at which patients first sat out of bed and were ready for discharge were recorded in all cases. All patients were followed-up by a telephone call on the day after their surgery and asked a standard set of questions about recall of peri-operative events, how they felt during the procedure, if they would have the procedure again with the same anaesthetic technique and if they liked administering their own sedation.

Pearson's correlation coefficient was calculated for the relationship between propofol dose and procedure duration, and Spearman's rank correlation was used for corresponding analysis involving surgical difficulty.⁷

Results

Patient and procedure details are shown in Table 1. Only two patients were male. Figure 1 shows the distribution for surgical difficulty and patient cooperation.

Cardiovascular stability was good (Fig. 2) and haemoglobin oxygen saturation remained 97% or higher in all cases. The deepest level of sedation reached by any patient corresponded to full eyelid closure with prompt response to verbal command.

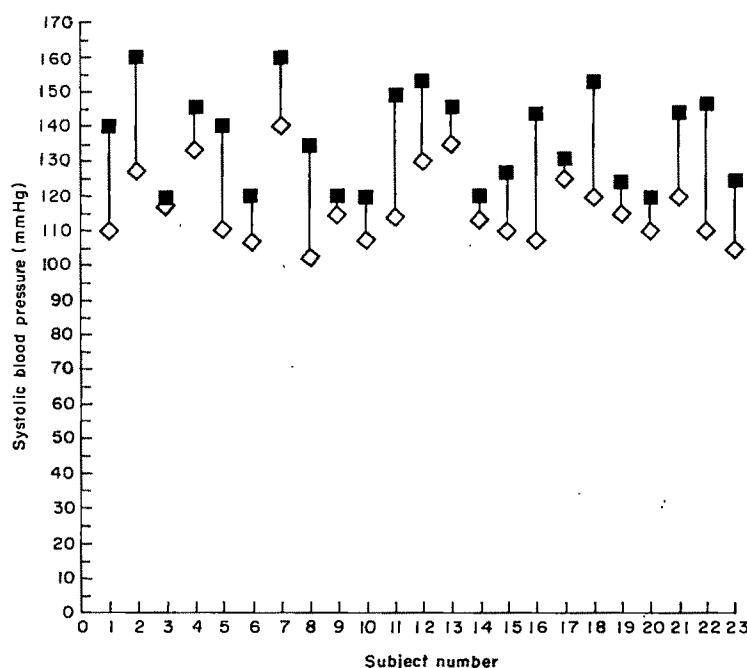


Fig. 2. Intra-operative systolic blood pressure range, for each patient. ■, maximum; ◇, minimum.

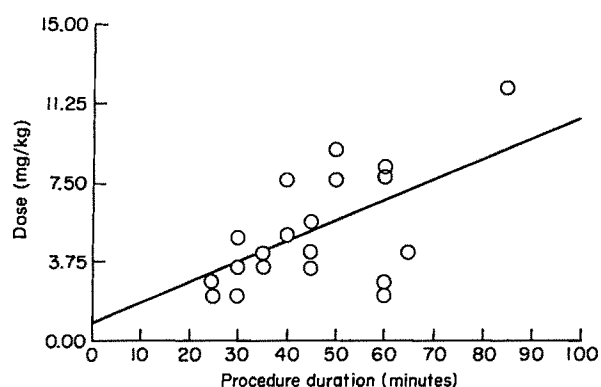


Fig. 3. Scatter plot of propofol dose (mg/kg) compared with procedure duration with line of best fit.

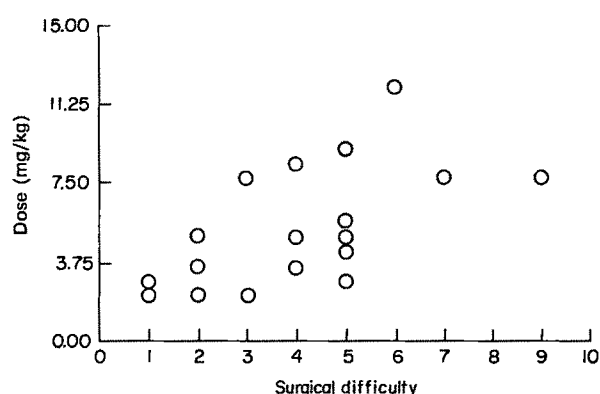


Fig. 4. Scatter plot of propofol dose (mg/kg) compared with surgical difficulty.

Significant correlation existed between propofol dose/kg body weight and both procedure length ($+0.587$, $p\ 0.003$) and surgical difficulty ($+0.55$, $p\ 0.01$) (Figs 3 and 4) despite large interpatient variation in the total number of attempts at self-administration (Table 2).

All patients at follow-up said they would have the same procedure using the PCS technique again. Subjective patient intra-operative feelings reported in terms of good, bad or indifferent were good in 22 cases and indifferent in the one remaining case. All patients stated they liked the self-administration of the drug.

Sixteen patients (70%) had amnesia for the extractions but none had amnesia for recovery events.

Discussion

Results indicate that PCS with propofol is a satisfactory method for sedation for the surgical extraction of third molars with local anaesthesia, a procedure that requires a significant level of sedation with a high margin of safety. Patient satisfaction was high and all patients not only felt comfortable with self-administration of the drug but stated that they liked to be in control of their own sedation.

Table 2. Summary data for number of patients attempts at self administration. Values expressed as mean (SD).

Successful attempts	6.4 (3.6)
Total attempts	34.8 (52.3)
Unsuccessful attempts	28.4 (49.5)

Cardiovascular stability was good and respiratory depression or other airway difficulties did not occur with the self-administration variables used in this study. There appeared to be no significant amnesia for the postoperative period; this is a significant advantage for day surgery.

The correlation between drug dose and both procedure length and surgical difficulty suggests that the pharmacokinetic properties of propofol and the patient-controlled technique may have combined to allow patients to titrate drug dose according to their own perceived needs against a background of interpatient variability in drug requirements. The technique therefore has potential for further investigation of interpatient variability in sedation requirements.

We believe that PCS with propofol will add a useful technique to the armamentarium for anaesthetic sedation. This can be applied to a wide range of procedures to provide safe, satisfactory sedation in a manner that meets informed patient expectations to a degree not previously attainable.

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Incremental spinal anaesthesia using a 32-gauge catheter

I. G. KESTIN AND N. W. GOODMAN

Summary

Incremental spinal anaesthesia using a 32-gauge intrathecal catheter was studied in 13 males scheduled for transurethral resection of the prostate or repair of inguinal hernia. The spinal technique failed in four patients. Dose-response curves were obtained in the remaining nine patients using increments of 0.5% plain bupivacaine. The spinal block was extended safely and reliably without cardiovascular instability. No patient had any postoperative headache and all catheters were removed intact.

Key words

*Anaesthetic techniques, regional; spinal.
Equipment, catheters; intrathecal.*

Continuous spinal anaesthesia using a subarachnoid catheter is a technique that was first reported in 1944,¹ but has not been widely used because only catheters of relatively large-bore have been available. A fine-bore 32-gauge catheter (TFX Medical) has recently been developed² and was used to investigate incremental spinal anaesthesia.

Methods

The project received ethics committee approval. Thirteen males scheduled for transurethral resection of the prostate or repair of inguinal hernia gave informed consent. The 32-gauge catheter was placed through a 26-gauge spinal needle (Becton-Dickinson), using an oblique paraspinous approach to L₃₋₄ with the patient lying on his left side. All catheters were placed by I.G.K. Approximately 3 cm of catheter, which has a single hole at the end, was left in the subarachnoid space.

The patient was then turned onto his back. Ten ml/kg of crystalloid was given intravenously before the first injection of 1–2 ml of 0.5% plain bupivacaine. The response was assessed after 15 minutes by the segmental loss of sensation to cold. The block was re-assessed every 3 minutes, and increments of 0.5–1 ml were given if two successive assessments were at the same segment. Increments were given until the height of the block was between T₈ and T₄. Heart rate and arterial blood pressure were recorded at 3-minute intervals.

As a separate study, 18 catheters (15 used and 3 new catheters from this and another study) were tested for strength by dead weight loading around cylindrical grips.

The accuracy of the loading was 0.1 g.

Statistical analyses were performed using linear regression and the *F*-test, with a significance level of 5%.

Results

The patients had a mean age of 70 (range 62–89) years, a mean weight of 73 (range 50–84) kg and mean height of 174 (range 160–187) cm. The spinal technique failed in four patients; twice the catheter kinked while it was threaded through the spinal needle; one of the catheter connectors leaked and in one patient the onset of block was slow and would not go above L₂.

Plots of height of block against dose of bupivacaine for the remaining nine patients are shown in Figure 1. There were significant linear regressions for the dose-response curves of all the patients, but the *F* values obtained from the log-dose response curves were 2–8 times higher than the corresponding *F* values obtained from the untransformed dose-response curves. There were no significant linear regressions of either the slopes or intercepts of the individual log-dose regression lines against the ages, weights, or heights of the patients. Haemodynamic stability was good during this slow induction of spinal anaesthesia; the greatest observed decrease in systolic blood pressure was from 200 mmHg to 140 mmHg and no patient needed vasopressors. The total time to extend the blocks to their final heights ranged from 27 minutes to 57 minutes.

All catheters were removed intact. No patient complained of a headache on direct questioning the next day.

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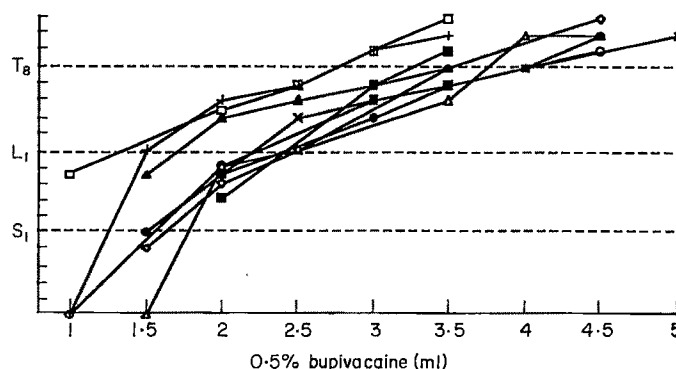


Fig. 1. Individual dose response curves, segmental height of block compared with ml of 0.5% bupivacaine, for incremental spinal anaesthesia through a 32-gauge catheter.

For convenience, the catheters were tested for strength in two batches. In the first batch 10 tests were performed. The mean strength was 1132 g (SD 59.1). In the second batch eight tests were performed. The mean strength was 1118.5 g (SD 119.9). There were two very low results (not included in the above figures) with failure at 705.1 g and at less than 650 g. The test was repeated for the two catheters that failed and adjacent sections failed at 1065.4 g and 1112.9 g, respectively.

Discussion

This study was carried out to gain some technical experience with the catheters before starting a trial in obstetric practice. An oblique paraspinous approach was chosen for two reasons: firstly, it may help to decrease the incidence of spinal headache,³ and, secondly, the oblique approach may make it easier to thread the catheter into the subarachnoid space. The catheter could be placed relatively easily using this method after the first few patients. Two catheters were kinked during insertion; this occurred in our first few patients and was caused by excessive pressure while threading through the spinal needle. Once the wire stiffener in the catheter is kinked, it is impossible to thread the catheter any further through the spinal needle or unthread the needle over the catheter.

All the dose-response curves had a positive slope, so the block could be extended as desired by giving increments. In contrast, studies using single doses of plain 0.5% bupivacaine given to patients lying flat have shown this technique is unreliable if a block above T₁₀ is required.^{4,5} The slopes of the curves were all similar and relatively shallow once the block was established, so it was possible to extend the block safely, without risk of sudden unpredictable extensions to high segmental levels. Most of the variability between patients was in the response to the initial dose; once the block was established, the rate of extension was similar in all the patients. These findings need confirmation in larger studies.

The catheters have an integral stiffener of wire along their length, incorporated into the design, after segments of the prototype catheters were left in some patients.² I.G.K.

found that the catheter can always be removed from the patient by gripping it firmly between thumb and forefinger and pulling gently along the line of insertion. No catheters have snapped when testing this grip on unused or removed catheters. There were two abnormally low values with no apparent explanation during the formal testing of 18, mostly used, catheters for strength. The wire stiffener may have been damaged during handling, because adjacent sections of the same catheters were of normal strength. Therefore, when taping the catheter in place, the catheter must not be allowed to kink at the skin, which might dangerously weaken the stiffener. This may be another advantage of the oblique approach to the subarachnoid space.

Our conclusions are that successful insertion of these catheters requires that they be handled carefully; success improved considerably with experience. The spinal block was extended safely and reliably after insertion using increments of 0.5% bupivacaine, without cardiovascular instability.

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Evaluation of local anaesthetic blockade of the lateral femoral cutaneous nerve

P. M. HOPKINS, F. R. ELLIS AND P. J. HALSALL

Summary

An assessment of local anaesthetic blockade of the lateral femoral cutaneous nerve using a standard technique was made. The rate of successful blockade was high, but the area of sensory loss was inconsistent between patients and was more anterior and distal than described in textbooks of anatomy.

Key words

Anaesthetic techniques, regional; lateral cutaneous nerve of thigh.

Local anaesthetic blockade of the lateral femoral cutaneous nerve has been proposed as an effective method of post-operative analgesia after hip surgery,¹ and is used to control lateral thigh pain in patients with meralgia paraesthetica, a complication of diabetes mellitus. We use blockade of this nerve in patients undergoing muscle biopsy from the vastus medialis as part of the so-called '3-in-1 femoral block',² and not by directly blocking the nerve. However, an evaluation of this block revealed that the lateral femoral cutaneous nerve was successfully blocked in only 67% of cases and in most of these the onset was delayed over 30 minutes compared to an onset time of less than 5 minutes for the femoral nerve itself.

The present study was undertaken to evaluate the area of sensory loss produced by the direct injection of local anaesthetic around the lateral femoral cutaneous nerve.

Method

Eighteen patients presenting for muscle biopsy for investigation of malignant hyperthermia susceptibility consented to be included in the study. The technique for lateral femoral cutaneous nerve blockade was similar to that described by Eriksson.⁴ A needle was inserted 2–3 cm medial and distal to the anterior superior iliac spine and at right angles to the skin. Ten ml of 0.5% prilocaine was then injected in a fan-wise manner through the fascia lata. A nerve stimulator was used in six of the patients in an attempt to improve nerve localisation.

The area of blockade was assessed at 5- and 10-minute intervals by loss of cold sensation using a swab soaked in

surgical spirit. This was immediately recorded on diagrams of the lower limb. A femoral nerve block was performed after 10 minutes using 0.5 ml/kg of 0.5% prilocaine.

Results

A successful block of the lateral femoral cutaneous nerve was recorded in 16 of the 18 patients; the two failures occurred in patients in whom the nerve stimulator was used. This was probably because stimulation of this sensory nerve proved very painful and was associated with a lot of patient movement. We therefore abandoned the nerve stimulator after its use in six patients. There was a great variation in the location and size of area of sensory loss even though there was a high success rate with this technique. The area was frequently 'pear-drop' shaped; the apex was over or distal to the greater trochanter and the sensory loss extended distally and somewhat anteriorly towards the knee. There was no region of the thigh that was consistently blocked in the 16 patients in whom a block was achieved. Figure 1 is a two-dimensional projection of the anterior and lateral aspects of the thigh showing the frequency of sensory loss in different regions.

Discussion

The standard technique of local anaesthetic blockade of the lateral femoral cutaneous nerve which was used in this study provided a high success rate. However, it has demonstrated that the area of skin supplied solely by this nerve lies more distally and anteriorly than might be inferred

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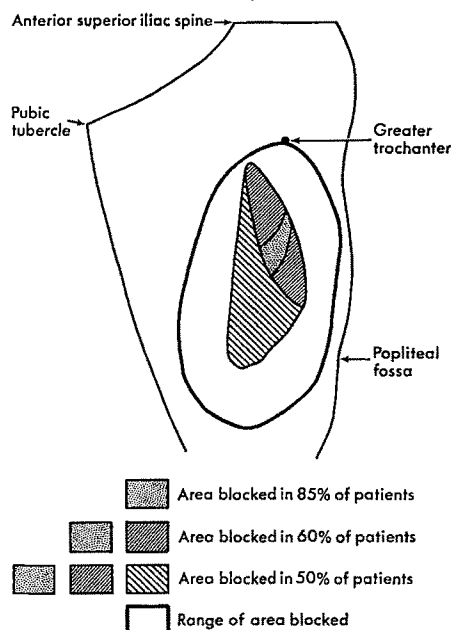


Fig. 1. Two-dimensional projection of the anterior and lateral aspects of the thigh showing the frequency of sensory loss in different regions.

from anatomy textbooks.³ Indeed, it is worthy of note that the lateral aspect of the thigh proximal to the greater trochanter was never blocked in this study. Perhaps the most clinically relevant feature is the great variation of the

area of sensory loss between individuals. This indicates that blockade of the nerve on its own is an unreliable method of providing local anaesthesia for any defined area, although its use for postoperative analgesia will reduce the requirements of systemic analgesics in some patients.¹ The variability of area of sensory loss demonstrated in this study probably reflects the variability in the overlap of supply by the different cutaneous nerves of the thigh; therefore blockade of the lateral femoral cutaneous nerve will continue to be required in combination with blockade of other nerves to provide regional anaesthesia of the thigh. In this respect, the standard technique of the block as used in the present study is more reliable, in terms of success rate and speed of onset, to the indirect approach to the lateral femoral cutaneous nerve using the 3:1 femoral technique.³

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Depth of extradural space and dural puncture

D. N. SUTTON AND S. P. K. LINTER

Summary

The records of 3011 obstetric extradural blocks were examined to determine the depth of the extradural space and the relative risk of dural puncture. The overall incidence of dural puncture was 0.7%. A disproportionate number of punctures occurred when the space was superficial (1.45%), with a risk factor three times greater than that associated with normal depth spaces (0.48%). Caution is advised when infiltrating with local anaesthetics before extradural procedures, and when initially introducing the extradural needle.

Key words

Anaesthetic techniques, regional; extradural.
Complications.

Continuous lumbar extradural analgesia is now an accepted and popular technique for administering pain relief in labour. One of the commoner complications is dural puncture, with its attendant risk of severe headache. Dural puncture in experienced hands can have an incidence as low as 0.2%;^{1,2} however, the overall rate with trainee anaesthetists is in the order of 1–3% and the highest is in the initial stages of training.^{1,2}

Identification of any risk factors associated with dural puncture would be of use to trainees in avoiding this complication. It was our impression that dural puncture often occurs at an unexpectedly short skin-to-extradural-space distance, and this hypothesis was investigated by retrospective review of 3011 consecutive cases.

Method

Lumbar extradural procedures were performed on 3011 women in labour by 26 anaesthetists in training over a 4-year period. The midline approach in a sitting position was used for all patients and the block was performed between the L₂₋₃ and L₄₋₅ spaces. Tuohy needles with 1 cm graduations were used and the extradural space identified by loss of resistance to air or saline. The skin to extradural space distance was recorded and the incidence and depth of dural puncture noted. Data were collected on standardised forms using tick boxes. Fisher's exact test was used to analyse data, with $p < 0.05$ considered statistically significant.

Results

The skin-to-extradural-space distance was recorded for 3011 procedures, and the distribution is shown in Figure 1.

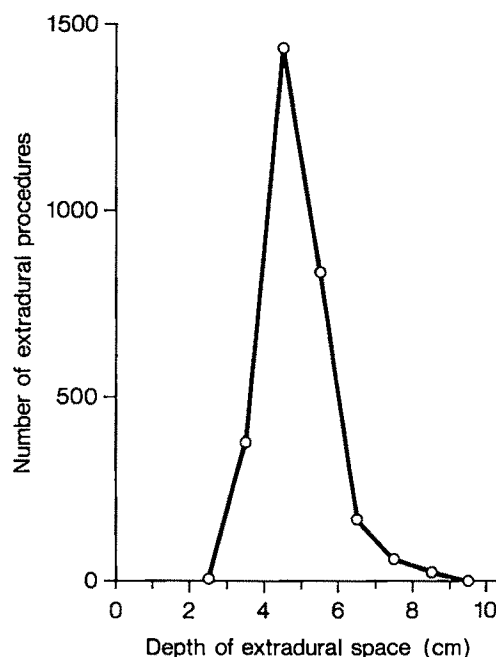


Fig. 1. Distribution of skin to extradural space distances in 3011 cases.

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Table 1. Relationship of depth of extradural space to number of extradural procedures, incidence of dural taps and dural puncture risk factors.

Depth of extradural space (cm)	Number of extradural procedures	Number of dural punctures	% of total extradural procedures	Risk factor
Shallow, 2-4	482 (16%)	7	1.45%	3
Normal, 4-6	2276 (76%)	11	0.48%	1
Deep, 6-9	253 (8%)	2	0.79%	1.6

Distances were placed in three groups for assessment of dural puncture risk; shallow 2-4 cm; normal 4-6 cm; deep 6-9 cm (Table 1). The normal group contained 76% of the total number of skin-to-extradural-space distances.

There were 20 dural punctures, an incidence of 0.7%. There was no association with the use of either air or saline, or with the vertebral interspace used. The dural punctures were compared to the depth of the extradural space and the incidence for each depth group calculated. A relative risk factor for dural puncture was then calculated, taking the lowest incidence (0.48%, normal depth 4-6 cm) as a factor of one (Table 1). The incidence of dural puncture at shallow depths was 1.45%, giving a risk factor of three ($p < 0.05$, 95% CI 1.16-7.65).

Discussion

The distribution of the depth of the extradural space agrees with other reports;^{3,4} 16% were shallow with a depth of 4 cm or less. However, there was an increased incidence of dural puncture at this depth (1.45%), and this was three times the incidence at a normal depth of 4-6 cm (0.48%), ($p < 0.05$). We believe that these findings are clinically significant and suggest that the extradural space is unexpectedly encountered when at a shallow depth. The shallow

extradural spaces can be within the range of a standard 21 or 23 gauge hypodermic needle (lengths 4 and 2.5 cm respectively), making dural puncture during initial infiltration with local anaesthetic possible if the needle is inserted to its full length.

Care must be taken when introducing an extradural needle, and it is suggested that the initial insertion should be to a depth of no more than 2 cm. Operators should be aware that there is a one-in-six chance of encountering the extradural space at a depth of 2-4 cm, with a correspondingly higher risk of dural puncture. Initial infiltration of local anaesthetic should be confined to superficial tissues, and the infiltrating needle should not be inserted to its full length.

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Echocardiographic assessment of the haemodynamic effects of propofol: a comparison with etomidate and thiopentone

A. GAUSS, H. HEINRICH AND O. H. G. WILDER-SMITH

Summary

The haemodynamic effects of propofol (2 mg/kg), etomidate (0.2 mg/kg) and thiopentone (4 mg/kg) were studied in 30 ASA 1 and 2 patients in whom anaesthesia had been induced with midazolam 0.1 mg/kg, fentanyl 5 µg/kg, vecuronium 0.1 mg/kg and atropine 10 µg/kg, and maintained with nitrous oxide in oxygen. Arterial pressure was measured directly and left ventricular diameters were determined by transoesophageal echocardiography. Systolic blood pressure after propofol and thiopentone and the end-systolic quotient (systolic pressure/end-systolic diameter), a measure of inotropy, decreased. Fractional shortening (end-diastolic – end-systolic diameter/end-diastolic diameter) decreased only in the thiopentone group. Diastolic blood pressure and end-diastolic diameter (a measure of preload) did not change in any of the groups, and the etomidate group showed no changes in the haemodynamic variables measured. Propofol shows simultaneous negative inotropy and afterload reduction, while thiopentone is exclusively negatively inotropic.

Key words

Anaesthetics, intravenous; propofol, thiopentone, etomidate.

Measurement techniques; transoesophageal echocardiography, electroencephalography.

Many studies have reported a reduction in arterial blood pressure after injection of the intravenous anaesthetic agent propofol. Reductions in blood pressure were more marked, and hypotensive reactions more frequent after propofol in comparison with etomidate¹ and thiopentone,^{2,3} but there is still no consensus in the literature why these reductions occur, be they negative inotropy, afterload reduction or both.

Almost all the patient studies to date, which were based on load-dependent variables, have differentiated poorly between the inotropic effects of propofol and its influence on myocardial loading factors.

The aim of our study was to demonstrate whether the decrease in arterial blood pressure after propofol is from negative inotropy and (or) changes in myocardial loading (pre/afterload), using a method considered to be relatively insensitive to load factors,⁴ namely inotropy measurements by transoesophageal echocardiography (TEE). In addition, we compared the haemodynamic effects of propofol with those of thiopentone and etomidate.

Methods

Thirty normotensive surgical or orthopaedic patients in ASA groups 1 and 2 were placed at random into either a

propofol ($n = 10$), thiopentone ($n = 10$) or etomidate ($n = 10$) group. None of the patients was receiving any concomitant cardiovascular medication, and all gave written informed consent to the study; the study itself was approved by the Ethics committee of the University of Ulm.

Each patient received orally 20 mg clorazepam as pre-medication one hour before the study. We injected atropine 10 µg/kg intravenously 5 minutes before induction, which was with midazolam 0.1 mg/kg and fentanyl 5 µg/kg. Vecuronium 0.1 mg/kg was given to facilitate tracheal intubation. Anaesthesia was continued after tracheal intubation, with controlled ventilation with $N_2O:O_2$ ($F_{IO_2} = 0.3$) to normocapnia ($P_{E'}CO_2 = 4.6$ – 6.0 kPa), (Capnometer Hewlett-Packard).

The radial artery was cannulated for blood pressure monitoring, and the transoesophageal echoscope (Varian 3400 R, Echoscope 3.5 MHz, Dasonics) inserted and positioned for a left-ventricular cross-section view of the middle of the papillary muscles. The M-mode cursor was set to the largest anteroposterior diameter and the resulting one-dimensional M-mode view displayed on the second monitor, together with arterial pressure curve, ECG and capnogram. All the data were recorded simultaneously on videotape.

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We recorded the left hemisphere EEG (F_3-A_1) (Lifescan EEG Monitor, Braun Melsungen AG) to control anaesthetic depth in 19 of the 30 patients (six in the propofol group, eight in the etomidate group and five in the thiopentone group) and obtained values for the median frequency (the frequency of the EEG power spectrum below which 50% of the total power is observed). In the first 11 patients no EEG was recorded.

We injected either propofol (2 mg/kg), etomidate (0.2 mg/kg) or thiopentone (4 mg/kg) over 60 seconds approximately 10 minutes after tracheal intubation, when haemodynamic and EEG steady-state had been achieved. We measured the following variables at end-expiration before injection (T0) and at one-minute intervals thereafter (T1–T10): heart rate and systolic/diastolic blood pressure; end-systolic and end-diastolic diameters* (in cm) of the left ventricle; and 50% edge frequency (in Hz) of the EEG.

We calculated the following derived values from the echocardiographic variables: end-systolic quotient = systolic pressure/end-systolic diameter;⁴ fractional shortening = end-diastolic diameter–end-systolic diameter/end-diastolic diameter. Evaluation of the echocardiograms, the measurement of diastolic and systolic pressures, and the determination of the heart rate took place on an electronic evaluation device (Cardio 200, Kontron). Only end-expiratory values were included. End-diastolic diameter was measured at the time of the R-wave on the ECG and the end-systolic diameter was taken as the smallest diameter in systole according to the trailing edge-leading edge method.⁵ All the haemodynamic measurements were made in the absence of surgical stimulation.

The Chi-squared test was used to check the comparability of the groups with regard to ASA grouping and sex; the Kruskal–Wallis test was used for age, height and weight. The median values and the 95% confidence limits at each time interval were calculated for each variable and each group. We checked the time course of the variables (T0–T10) for significant changes using the Friedman test, and because we used multiple tests on the same data for the same question, the Bonferroni–Holm correction for error probability was used. Only the corrected error probabilities were included in our evaluation. Comparison between the groups at time T0 and times T1–T10 was performed by the Kruskal–Wallis test. Here the minima (systolic and diastolic pressure, end-systolic quotient, fractional shortening and end-diastolic diameter) or maximum (heart rate) of each patient's time course was used for the comparison and the significance level was taken as $p < 0.05$ for all tests.

Results

The demographic data are shown in Table 1; there were no significant differences in age, weight, height, sex or ASA status between the three groups. Tables 2 and 3 show the medians of the measured and derived haemodynamic variables, together with the median values of the median frequency of the EEG.

Arterial systolic blood pressure only decreased after thiopentone and propofol, but there was no change with etomidate; there were no significant changes in the diastolic pressure. Heart rate increased in the first few minutes after

Table 1. Demographic data of patients, median and (range).

	Propofol	Etomidate	Thiopentone
Number of patients	10	10	10
Age; years	22 (19–34)	27.5 (18–45)	25.5 (19–46)
Weight; kg	70 (44–90)	75 (55–93)	69 (57–87)
Height; cm	177 (144–193)	175.5 (165–190)	178 (158–183)
Sex: Male	9	9	8
Female	1	1	2
ASA group 1	9	10	8
2	1		2

the injection of propofol or thiopentone, but not etomidate (propofol 23%, thiopentone 11% on average). There was no change in end-diastolic diameter in any group. The end-systolic quotient decreased after propofol and thiopentone (about 20% on average for both), but there was no change in this variable with etomidate (Fig. 1).

Only thiopentone reduced fractional shortening (about 14% on average) (Fig. 2). There was no statistically significant difference between the groups at time T0, before injection of the induction agent, nor in the depth of anaesthesia as measured by the median frequency of the EEG or haemodynamic variables. There were no significant differences between the groups in haemodynamic variables in relation to time.

Discussion

We used the end-systolic quotient as our inotropy variable instead of the slope of the systolic pressure-diameter relationship because the pressure manipulation necessary to determine end-systolic pressure volume or pressure-diameter relationships is inapplicable to the bolus injection of anaesthetic agents. Steady-state drug concentrations are not achieved, nor is it easy, because of different pharmacokinetics and dynamics, to predict times of comparable haemodynamic effect at which such a manipulation would logically have to occur. We could only determine peak systolic pressure with the catheter in the radial artery (but not end-systolic pressure). However, peak systolic pressure is also related linearly to end-systolic dimension;⁶ the end-systolic quotient is a variable derived⁴ from the end-systolic pressure volume or pressure diameter relationship.^{7–10} An increase in inotropy is associated with an increase in end-systolic quotient and the converse also applies. The end-systolic quotient shows a dependence on afterload (end-systolic quotient falls with decreasing afterload and rises with increasing afterload), but is insensitive to changes in preload. Nevertheless, increases in inotropy remain demonstrable even in the face of extreme afterload reduction.⁴

We determined fractional shortening as an additional confirmatory factor for changes in inotropy. This is helpful because fractional shortening shows, in addition to a dependency on preload, a marked dependence on afterload, but in the opposite direction to end-systolic quotient (reduction of fractional shortening with rising afterload, increase of fractional shortening with falling afterload). Thus simultaneous changes of inotropy and afterload can

*as a measure of preload.

Table 2. Measured haemodynamic variables (median values) (T0–T10).

	T0	T1	T2	T3	T4	T5	T6	T7	T8	T9	T10
<i>Systolic arterial pressure (mmHg)</i>											
Propofol*	101 (92–117)	96 (79–103)	88 (82–98)	87 (80–101)	90 (86–94)	90 (82–103)	89 (81–104)	89 (76–103)	91 (80–105)	92 (77–107)	92 (74–104)
Etomidate	114 (83–126)	119 (85–124)	115 (81–119)	109 (76–120)	114 (80–121)	109 (82–129)	108 (82–136)	116 (83–134)	113 (79–141)	109 (80–141)	115 (84–142)
Thiopentone*	106 (76–131)	101 (74–119)	94 (73–122)	94 (62–127)	99 (82–128)	97 (69–121)	100 (71–119)	97 (68–121)	97 (71–121)	93 (77–123)	95 (78–122)
<i>Diastolic arterial pressure (mmHg)</i>											
Propofol	56 (54–69)	55 (41–69)	52 (47–62)	53 (46–67)	55 (46–63)	52 (44–64)	53 (47–63)	58 (46–66)	54 (43–66)	55 (44–67)	51 (45–63)
Etomidate	64 (47–71)	64 (54–75)	63 (54–73)	57 (50–70)	64 (52–72)	61 (54–79)	64 (52–86)	67 (51–82)	65 (45–88)	62 (45–85)	67 (49–89)
Thiopentone	58 (50–81)	59 (45–69)	59 (43–70)	61 (31–72)	62 (50–73)	61 (51–73)	58 (49–72)	58 (38–74)	59 (44–74)	57 (43–71)	57 (45–70)
<i>Heart rate (per minute)</i>											
Propofol***	65 (60–93)	80 (69–94)	73 (64–87)	70 (60–84)	70 (60–86)	67 (59–84)	66 (61–85)	68 (60–84)	66 (59–82)	67 (60–83)	65 (60–81)
Etomidate	71 (62–82)	72 (66–82)	71 (65–81)	69 (65–86)	71 (64–86)	70 (63–89)	69 (61–87)	70 (62–86)	71 (61–85)	71 (63–88)	71 (61–83)
Thiopentone***	73 (62–79)	78 (66–84)	81 (73–91)	79 (72–84)	80 (70–84)	78 (68–86)	78 (68–89)	77 (67–86)	77 (66–85)	77 (65–85)	76 (60–83)
<i>End-diastolic diameter (cm)</i>											
Propofol	5.00 (3.61–6.77)	5.12 (3.57–6.83)	5.15 (3.71–6.87)	5.24 (3.76–6.84)	5.23 (3.8–6.8)	5.21 (3.69–6.81)	5.27 (3.82–6.87)	5.20 (3.66–6.87)	5.32 (3.76–6.78)	5.07 (3.79–6.81)	5.22 (3.76–6.8)
Etomidate	5.00 (4.68–5.63)	4.91 (4.67–5.51)	4.94 (4.66–5.53)	4.97 (4.52–5.48)	5.02 (4.58–5.38)	4.97 (4.7–5.42)	5.00 (4.65–5.41)	4.99 (4.63–5.67)	4.95 (4.54–5.42)	4.93 (4.44–5.41)	5.03 (4.56–5.36)
Thiopentone	4.60 (3.45–5.58)	4.76 (3.39–5.78)	4.62 (3.43–5.85)	4.55 (3.2–5.96)	4.49 (3.31–5.83)	4.55 (3.23–5.88)	4.50 (3.39–5.6)	4.41 (3.35–5.55)	4.46 (3.38–5.66)	4.48 (3.35–5.59)	4.37 (3.42–5.65)

Significant changes over the time course within a group:

* $p < 0.05$; *** $p < 0.001$ (Friedmann-test).

95% confidence limits in parentheses.

be separated as long as the preload (end-diastolic diameter) remains unchanged.

Transoesophageal echocardiography in the conscious patient would have been unreasonable, so we chose a design in which we tested the effects of the intravenous

anaesthetics during basal anaesthesia and with established monitoring. Performing these measurements during steady-state basal anaesthesia (of equal EEG depth)¹¹ has the additional advantage of eliminating errors from differences in pre-anaesthetic sympathetic tone or in ventilation.

Table 3. Derived haemodynamic variables and the time course of the 50% edge of the EEG (median values) (T0–T10).

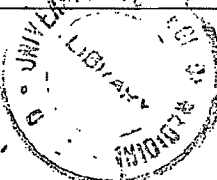
	T0	T1	T2	T3	T4	T5	T6	T7	T8	T9	T10
<i>End-systolic quotient (mmHg/cm)</i>											
Propofol**	29.4 (20.7–37.9)	23.6 (18.5–34.2)	25.0 (18.9–33.4)	24.8 (18.5–32.5)	23.8 (18.7–32.9)	26.8 (16.9–33.7)	26.8 (18.2–34.0)	25.7 (19.2–31.8)	26.2 (19.4–33.3)	24.8 (20.4–34.5)	25.3 (18.2–35.8)
Etomidate	31.2 (27.2–33.1)	31.2 (24.1–35.3)	30.7 (24.9–33.0)	30.3 (23.9–33.1)	30.1 (24.2–34.0)	30.1 (24.7–33.2)	30.5 (25.5–35.5)	31.4 (25.7–33.8)	31.1 (25.1–35.1)	29.9 (23.3–35.4)	30.8 (26.6–35.7)
Thiopentone**	31.9 (23.7–50.6)	29.2 (20.4–42.1)	26.5 (20.0–41.3)	25.1 (17.9–46.5)	29.7 (23.7–48.3)	30.1 (20.9–47.4)	29.6 (21.4–47.3)	30.1 (20.5–47.9)	30.0 (20.9–45.6)	30.0 (23.0–43.5)	30.0 (22.4–42.4)
<i>Fractional shortening</i>											
Propofol	0.290 (0.24–0.36)	0.285 (0.23–0.37)	0.285 (0.25–0.36)	0.265 (0.24–0.36)	0.290 (0.24–0.39)	0.285 (0.25–0.38)	0.315 (0.25–0.35)	0.295 (0.26–0.37)	0.290 (0.25–0.38)	0.310 (0.26–0.36)	0.295 (0.25–0.36)
Etomidate	0.290 (0.26–0.33)	0.280 (0.26–0.32)	0.265 (0.23–0.32)	0.275 (0.22–0.3)	0.280 (0.21–0.3)	0.280 (0.23–0.32)	0.290 (0.23–0.36)	0.295 (0.21–0.31)	0.285 (0.22–0.32)	0.290 (0.22–0.31)	0.275 (0.22–0.33)
Thiopentone**	0.280 (0.23–0.38)	0.240 (0.21–0.36)	0.245 (0.2–0.33)	0.255 (0.19–0.35)	0.280 (0.2–0.35)	0.270 (0.21–0.36)	0.280 (0.22–0.35)	0.285 (0.19–0.35)	0.300 (0.23–0.34)	0.265 (0.22–0.37)	0.260 (0.2–0.37)
<i>EEG (50% edge)</i>											
Propofol	13 (3–16)	6 (1–11)	3 (1–11)	2 (1–8)	4 (0.5–9)	4 (1–10)	6 (4–11)	6 (3–10)	8 (5–11)	8 (4–11)	8 (6–12)
Etomidate	9 (1–12)	8.5 (1–11)	3 (2–8)	4 (2–5)	5 (3–8)	6.5 (3–9)	7.5 (3–10)	8.5 (4–10)	8.5 (3–10)	8.5 (2–11)	8 (3–11)
Thiopentone	7 (2–12)	9 (2–13)	3 (1–8)	4 (2–9)	7 (2–9)	9 (3–10)	9 (6–10)	10 (8–10)	9 (8–11)	10 (7–11)	9 (8–11)

Significant changes over the time course within a group:

** $p < 0.01$ (Friedmann-test).

In brackets () = 95% confidence limits.

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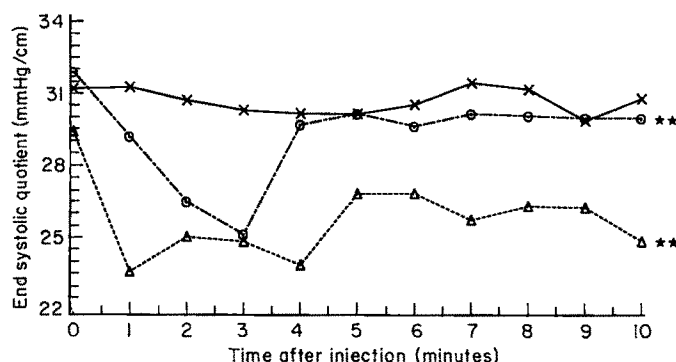


Fig. 1. Median course of the end-systolic quotient after etomidate 0.2 mg/kg (x—x), propofol 2 mg/kg (Δ—Δ), or thiopentone 4 mg/kg (○—○); ** $p < 0.01$.

We chose midazolam, fentanyl and vecuronium in order to minimise the influence of this basal anaesthesia on the effects of the intravenous anaesthetic agents. These drugs have minimal effects on the cardiovascular system.^{12,13} Interactions for the combination of propofol and fentanyl have been described.¹⁴ Major cardiovascular changes due only to the transition at induction from a conscious to an unconscious state can be avoided with this scheme. We were able to demonstrate similar basal conditions for all three groups both with regard to depth of anaesthesia and haemodynamic variables.

Equipotent doses of induction agents should be studied in the comparison of haemodynamic effects. Compared with MAC (volatile anaesthetics) and MIR (intravenous anaesthetics by infusion) no equivalent exists for the bolus injection of induction agents. An equipotency ratio of 1:1.6 is quoted for propofol and thiopentone given as bolus injections.¹⁵ Edelist¹⁶ found a higher incidence of supplementary injection for thiopentone than for propofol when using this dose ratio, which resulted in a final dose ratio of 1:2.2. Ulsamer, Doenicke and Laschat¹⁷ used an average of 1.8 mg/kg propofol and 0.2 mg/kg etomidate after lormetazepam and fentanyl. Induction doses of intravenous anaesthetics based on hypnotic potency alone usually differ from those found necessary by clinical experience. Our doses take into account the relevant literature and clinical experience.

We found no significant change in fractional shortening in our study after propofol, and because the end-systolic quotient decreased at the same time, we conclude that

propofol has both negative inotropic and afterload reducing properties. The injection of thiopentone resulted in a simultaneous reduction of end-systolic quotient and fractional shortening, which indicates a mainly negative inotropic effect. Etomidate was devoid of significant haemodynamic effects.

The available literature reports varying effects of propofol on inotropism and afterload, and differing mechanisms for the decrease in blood pressure. Comparison of studies is difficult because of differences in patient selection, premedication, concomitant medication, anaesthetic technique, doses, monitoring techniques and statistical analysis.

To date, only very few authors have attempted direct measurement of propofol's effect on inotropism. Brüssel *et al.*¹⁸ found a significant reduction in dogs in the so-called left ventricular forces (dF/dt) together with a reduction of cardiac output, but with unchanged peripheral resistance and cardiac filling pressures. The authors postulated a negative inotropic effect for propofol in contrast to etomidate. This agrees only in part with our results: if propofol only had negative inotropic properties, we would have expected a decrease in fractional shortening. The latter was only preserved by the additional lowering of afterload in our study.

Kling *et al.*¹⁹ reported a 28% decrease of left ventricular dp/dt with only a minor reduction in left ventricular end-diastolic pressure, in the context of an aortocoronary bypass operation. This would support a negative inotropic effect of propofol. The maximum left ventricular dp/dt is of only limited use, however, as an inotropism variable

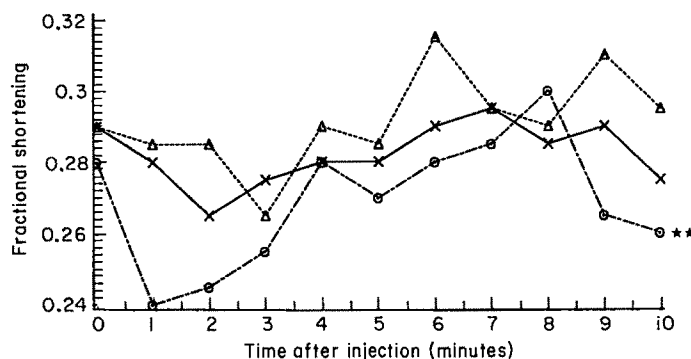


Fig. 2. Median course of the fractional shortening after etomidate 0.2 mg/kg (x—x), propofol 2 mg/kg (Δ—Δ), or thiopentone 4 mg/kg (○—○); ** $p < 0.01$.

because of its dependence on pre- and afterload. The same authors studied the effects of propofol during extracorporeal circulation and found, in contrast to midazolam, a significant reduction of mean arterial pressure at constant machine flow. They thus attributed blood pressure decreases after propofol mainly to peripheral vasodilatation.

Studies in cats have neither demonstrated ganglion blocking nor alpha-receptor blocking properties for propofol.²⁰ The decrease in peripheral resistance has until now been considered to be a direct action on vascular smooth muscle. Fractional shortening remained constant during our study which is against a purely peripheral vasodilatory action of propofol; an isolated reduction in afterload should have resulted in an increase of fractional shortening.

Patient studies on the haemodynamic effects of propofol using pulmonary artery catheters are only able to make indirect statements about myocardial contractility; the results of these studies are also variable.

Van Aken *et al.*²¹ found a reduction in cardiac output with unchanged peripheral resistance and filling pressures, which would suggest that propofol has a negative inotropic action. Those studies which found a reduction in mean arterial pressure together with a reduction in cardiac output and peripheral resistance²²⁻²⁵ would indicate that, in agreement with our study, propofol combines negative inotropism with reduction in afterload. An unchanged cardiac output or cardiac index in combination with stable filling pressures after propofol does not necessarily mean a lack of myocardial depression;^{26,27} an isolated reduction in afterload would cause an increase in cardiac output.

We used nitrous oxide in our study, as in others.^{21,22,24,25} It is difficult to say whether the combination of nitrous oxide with propofol attenuated the reduction in peripheral resistance, or even prevented it altogether. Negative inotropism, apart from a vasoconstricting effect, is also postulated for nitrous oxide,²⁸ but we do not know of any studies which examine the possible potentiation of negative inotropic effects from the interaction between nitrous oxide and thiopentone or propofol.

The preload, a further determinant of myocardial function, remained constant with all three induction agents in our study. This is important, because changes in preload can alter stroke volume i.e. fractional shortening. Most other workers also reported no relevant changes in central venous or pulmonary capillary wedge pressure after propofol.^{22,23,26,29} Lepage *et al.*³⁰ were the only ones to report a decrease in preload in propofol.

Thiopentone resulted in a decrease in end-systolic quotient and fractional shortening in our study. This agrees with most reports in the literature, which describe thiopentone as having a predominantly negative inotropic mode of action, with a constant³¹ or even slightly rising peripheral resistance,³² but there are studies which report a reduction of peripheral resistance together with unchanged cardiac output for thiopentone.³³ Some of the older literature discusses a reduction of venous return as the cause of the decrease in cardiac output.^{34,35} Our study, with its constant preload, provides no support for such a theory.

The stable haemodynamics we found with etomidate confirm other authors' findings.^{36,37} However, in an animal model Kissin *et al.*³⁸ found a reduction in peak tension development and postulated a dose-dependent negative

inotropism for etomidate. A decrease in mean arterial pressure, together with a reduction in cardiac output without alteration of peripheral resistance, was described by Criado *et al.*³⁹ This was taken to signify a negative inotropic effect of etomidate, and a reduction of left ventricular dp/dt with unchanged filling pressures has also been described.^{40,41} A minor degree of negative inotropism was therefore discussed.⁴¹ In contrast we were unable to demonstrate a negative inotropic action of etomidate; whether this is solely a problem of dose cannot be answered with certainty.

We found an increase in heart rate after propofol, although there was no concomitant surgical stimulation. Few authors describe such an increase;^{29,42} most observed a strikingly stable heart rate.^{19,23-25,43} Cullen *et al.*⁴⁴ report that for propofol, lower heart rates are possible with intact baroreflex sensitivity despite low blood pressures than for other intravenous anaesthetics.

The increase in pulse rates in our study can be partially explained by the atropine which was given before induction. We considered this necessary because of the danger of bradycardia associated with the combination of fentanyl and vecuronium. In addition we were worried by the possibility of vagal reaction to the introduction of the oesophageal echoscope. A further explanation is the fact that we examined young patients with a healthy cardiovascular system, without comedication such as beta-blockers, and, most probably, with intact baroreflex function.

We found no significant differences between the groups with regard to blood pressure, heart rate, end-systolic quotient or fractional shortening. This is most probably the result of the small numbers of patients. Normal values are not yet available for the end-systolic quotient,⁸ and only intra-individual comparisons can be expected to yield significant results for small samples.

In conclusion, it proved feasible to demonstrate the effect of propofol and other induction agents on inotropism and afterload by relatively noninvasive means: namely by end-systolic quotient and fractional shortening from transoesophageal echocardiographic data and the peripheral blood pressure. In particular, we were able to demonstrate a simultaneous reduction in inotropism and afterload by propofol.

The simultaneous reduction in afterload following propofol results in a maintenance of fractional shortening, despite its negative inotropic effects. Thus propofol appears more favourable than thiopentone for patients with heart failure and increased peripheral resistance. Incremental doses are recommended for patients with cardiac failure and relative hypovolaemia receiving diuretic therapy or hypovolaemia from other causes: serious hypotension can result from bolus injection, as a result of the combined effects of negative inotropy and afterload reduction. Particular caution should be taken in patients with stenosis of the main stem of the coronary artery or significant carotid artery stenosis; we prefer etomidate for these patients because of its relative lack of haemodynamic side effects.

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CASE REPORT

The anaesthetic management of congenital tracheal stenosis

P. M. MURPHY AND A. LLOYD-THOMAS

Summary

Congenital tracheal stenosis is a rare and potentially lethal condition. Two case reports are presented which demonstrate the problems encountered in the anaesthetic management of this condition.

Key words

Lung; tracheal stenosis.

Anaesthetic techniques.

Congenital tracheal stenosis (stovepipe trachea) is an intrinsic narrowing of the lumen by rigid and nondistensible tracheal walls.¹ The management of this condition is difficult and until recently it was thought to be almost uniformly fatal. However, over the last decade there have been reports in the literature of successful surgical treatment using a variety of techniques. Early recognition of this condition is important as prompt treatment is likely to improve outcome.

Case histories

Case 1

This patient, born at term, was apnoeic and required immediate tracheal intubation and assisted ventilation. He continued to have respiratory difficulties despite establishing spontaneous respiration at 15 minutes and a chest X ray showed a hypoplastic right lung with displacement of mediastinal contents to that side, and lower cervical and upper thoracic hemivertebrae.

Bronchoscopy on the second day of life showed marked distal tracheal narrowing. He was weaned from mechanical ventilation and discharged home at the age of 3 weeks. He had five further admissions to hospital due to chest infections over the next 4 months and was managed conservatively on each occasion. Cardiac catheterisation demonstrated a pulmonary artery sling but otherwise normal cardiac anatomy. He presented acutely with upper airway obstruction at the age of 6 months and required tracheal intubation and mechanical ventilation. Bronchoscopy confirmed the stenotic tracheal segment and a barium swallow showed a vascular ring. The anomalous

left pulmonary artery was corrected and several attempts made at endoscopic dilatation of the trachea, but it was impossible to wean him from respiratory support. Mechanical ventilation became increasingly difficult and he suffered two hypoxic cardiac arrests. He was referred to The Hospital for Sick Children, London, at the age of 11 months after a tracheobronchogram showed that the stricture extended from the midtrachea to one centimetre above the carina. A computerised axial tomographic scan (CT scan) confirmed a midtracheal stenosis and it was decided to perform a tracheoplasty with a pericardial patch.

Anaesthesia was induced with fentanyl 5 µg/kg and diazepam 0.2 mg/kg intravenously and maintained with 0–1% halothane and 50% nitrous oxide in oxygen. Pancuronium 0.1 mg/kg provided muscle relaxation. The tracheal tube (3.5 mm internal diameter) was repositioned above the level of the stenosis. Mechanical ventilation using a Servo 900 D ventilator remained difficult and tidal volumes of 10 ml/kg, a ventilatory rate of 25 breaths/minute and inflation pressures of 5.5–6.0 kPa were required to inflate the lungs adequately. Humidification of inspired gases was achieved with a Bennett cascade humidifier. Cardiopulmonary bypass was established and the patient cooled to 18°C before the circulation was arrested. A longitudinal tracheal incision was made from above to just below the stenotic segment and the defect closed with a piece of porcine pericardium. The nasotracheal tube was positioned below the level of the tracheoplasty under direct vision. There was no leak from the repair on reinstitution of positive pressure ventilation and inflation pressures of 3–4 kPa resulted in adequate ventilation. However, he developed streptococcal septicaemia during the first post-

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operative night, and failed to respond to antibiotics, expansion of the circulating volume or inotropic drugs. A postmortem examination confirmed that the operation had been technically successful but revealed multiple hepatic, splenic and pulmonary septic foci.

Case 2

This female, term infant, had an imperforate anus, a recto-vaginal fistula, vesico-ureteric reflux and mild inspiratory stridor. She underwent three operations (at birth, 3 weeks and 7 weeks); on each occasion only a size 2.5 mm oral tracheal tube could be passed and was a tight fit. She was presumed to have subglottic narrowing but as she was clinically well this was not investigated further. She presented at 7 months with marked biphasic stridor, sternal recession and cyanosis. Acute laryngotracheobronchitis was suspected. Her upper airway obstruction deteriorated and she underwent diagnostic bronchoscopy and tracheal intubation. Anaesthesia was induced with halothane in oxygen and topical lignocaine 3 mg/kg was applied to the larynx. However, it proved impossible to pass a size 2.5 mm tracheal tube or bronchoscope into the distal trachea. The appearances were typical of a congenital tracheal stenosis with complete cartilaginous rings and a luminal diameter of 1 mm. A size 2.5 mm nasotracheal tube was left *in situ*, cut to ensure that its tip was proximal to the stenosis. She required inflation pressures of up to 8 kPa despite neuromuscular blockade with a vecuronium infusion 80 µg/kg/hour during mechanical ventilation with a Servo 900 C ventilator. A CT scan showed tracheal stenosis with no bronchial involvement. Mechanical ventilation was easier over the next 4 days and she was allowed to breathe spontaneously with continuous positive airway pressure (CPAP). A barium swallow suggested the presence of a vascular ring and angiography showed this to be a pulmonary artery sling. It was decided to perform a tracheoplasty and to reconstruct the aberrant pulmonary artery.

Anaesthesia was maintained with 50% nitrous oxide and 0–1% halothane in oxygen, and boluses of fentanyl (total 10 µg/kg). Muscle relaxation was established with pancuronium 0.1 mg/kg. A Servo 900 D ventilator delivered tidal volumes of 10 ml/kg at a rate of 30 breaths/minute. Inspired gases were humidified with a Bennett cascade humidifier. Initial inflation pressures were 7–8 kPa. The patient was cooled to 28°C after institution of cardiopulmonary bypass. The pulmonary artery sling was corrected uneventfully. The stenotic tracheal segment, with complete cartilaginous rings and measuring 2 cm in length, was then excised and a primary anastomosis was formed. A residual small anterior defect was repaired with a vascularised flap of autologous pericardium. No leak was demonstrated from the anastomosis and on resumption of positive pressure ventilation inflation pressures were 3–4 kPa.

She was weaned to spontaneous ventilation with CPAP over 36 hours postoperatively. However, her respiratory status then began to deteriorate with increasing chest recession, carbon dioxide retention and high inflation pressures. An emergency bronchoscopy showed an inflamed lower trachea tapering towards the site of the anastomosis which was mildly oedematous; the tracheal lumen was occluded by a plug of mucus. She improved after removal of the plug and her trachea was extubated 4 days later. A nasal prong

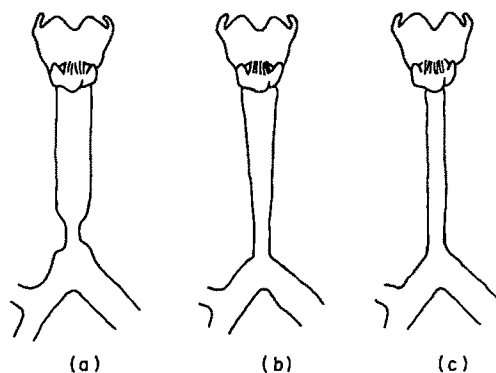


Fig. 1. Diagram of the three types of congenital tracheal stenosis: (a) segmental stenosis (b) funnel-shaped stenosis (c) generalised stenosis.

was used to provide CPAP for a further 5 days. She redeveloped biphasic stridor and required reintubation and ventilation because of increasing respiratory distress. A size 3.0 bronchoscope could not be passed further than the midtrachea. The distal trachea, carina and right main bronchus were dilated forcibly with an angioplasty balloon which improved ventilation slightly. She was weaned from the ventilator over the next 6 days and her trachea was extubated.

She was discharged 11 days later (39 days after surgery) with mild residual stridor. She has remained well in the succeeding 8 months.

Discussion

There are three types of congenital tracheal stenosis: segmental, funnel-shaped and generalised hypoplasia² (Fig. 1). All three types are characterised by complete, or near complete, tracheal cartilaginous rings that lack the normal structure of the posterior membranous trachea.³ The condition presents usually in infancy with signs of airway obstruction, although it may present in adults as a chance finding.^{4,5} Stridor, which is often biphasic, is the commonest sign and may be accompanied by other signs of airway obstruction such as chest retraction and use of secondary muscles of respiration.¹ There is often a history of frequent chest infections and these may result in fatal respiratory failure as critical airway obstruction may be precipitated by small amounts of secretions or oedema. Associated anomalies of the tracheobronchial tree, the cardiovascular system, the skeleton or the oesophagus are common.¹ Both of our patients had associated anomalies. A vascular ring, which has been reported to occur in 50% of patients in a previous series,⁶ may cause diagnostic confusion and resulted in inappropriate initial management in Case 1. The diagnosis of congenital tracheal stenosis must always be considered in any child who presents with recurrent respiratory infections and airway obstruction. The presenting features, associated anomalies, bronchoscopic findings and clinical course in Case 1 should have suggested the diagnosis of congenital tracheal stenosis with complete cartilaginous rings, and earlier referral may have resulted in a more successful outcome.

Anaesthesia for bronchoscopy

Anaesthesia for diagnostic bronchoscopy is a potentially hazardous procedure because of the danger of precipitating total airway occlusion by traumatising the stenosis.

Facilities for immediate thoracotomy and institution of cardiopulmonary bypass (CPB) should be available if the diagnosis is suspected and the airway is already severely compromised. Anticholinergic premedication helps prevent serious bradyarrhythmias on induction of anaesthesia and prevents excessive secretions during manipulation of the airways. However, drying of the mucosa may make it more susceptible to the trauma of a tracheal tube if the tube contacts the stenotic segment. Sedative premedication must be avoided. An inhalational induction of anaesthesia with halothane in oxygen should be used. Laryngoscopy should be performed under deep inhalational anaesthesia and the larynx and trachea sprayed with lignocaine 10% (3 mg/kg). The tracheal tube must be positioned at least one centimetre proximal to the stenosis to avoid traumatic oedema or total obstruction at the upper end of the stenosis. Monitoring of oxygen saturation, electrocardiogram, blood pressure and temperature are essential. Anaesthesia is deepened with oxygen and halothane and the tracheal tube is exchanged for a Stortz bronchoscope; spontaneous ventilation of the anaesthetic gas mixture is continued via the bronchoscope. Passage of the bronchoscope through the stenosis is almost invariably impossible and should not be attempted because of the risk of causing mucosal oedema and precipitating total airway obstruction.

The trachea may or may not be extubated at the end of the bronchoscopy depending on the findings and the ventilatory status of the patient. Positive pressure ventilation may be required before a definitive surgical procedure, and is likely to be difficult. The child should undergo nasotracheal intubation if mechanical ventilation is necessary; again the tip of the tracheal tube should be positioned one centimetre above the narrowing. Passage of suction catheters should be controlled and kept to a minimum to avoid further trauma to the stenosis. Regular physiotherapy, humidification of inspired gases and treatment of proven chest infections with antibiotics are all essential in the intervening period. Tracheostomy is contraindicated as an initial procedure because the distal tracheal lumen may be too narrow and it may compromise a future resection site.⁷

Surgical repair

The type of operation depends on the position and length of the stenosis. Resection and end-to-end anastomosis is feasible in short and localised segments of tracheal stenosis.⁸ Longer segments can be managed by tracheoplasty utilising autologous pericardium⁹ or costal cartilage grafts¹⁰ (Fig. 2). The pericardial patch becomes incorporated into the anterior tracheal wall, and fibroses and calcifies. Forceful endoscopic dilatation without surgical repair is a desperate procedure with a high incidence of serious complications.¹¹

Anaesthesia for surgical repair

The airway is secured as described for bronchoscopy. High ventilatory pressures are likely to be needed, with high levels of positive end-expiratory pressure (PEEP). A long-acting muscle relaxant may be administered provided that the lungs can be ventilated. The position of the tip of the tracheal tube can be assessed using a fiberoptic bronchoscope. Monitoring should include pulse oximetry, capnography and airway pressure. An arterial line should be

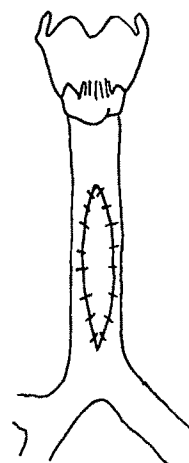


Fig. 2. Tracheoplasty. The stenotic segment of trachea is incised longitudinally and the anterior tracheal wall is reconstructed with a graft.

inserted for blood gas sampling. A central venous line via the femoral vein is advisable; the internal jugular and subclavian veins should be avoided because cannulation may interfere with the surgical field, particularly if a tracheostomy is fashioned at the end of the operation to stent the trachea. Humidification of inspired gases is important. Peri-operative parenteral steroids are controversial but topical steroids are not of any proven value.

Management of ventilation

Ventilatory management has been approached in a number of different ways. The methods available are standard positive pressure ventilation, high frequency jet ventilation (HFJV), cardiopulmonary bypass (CPB), or a combination of these. Maintenance of spontaneous ventilation throughout the procedure is unsuitable.

A variety of techniques utilising standard positive pressure ventilation have been used to ensure adequate ventilation. Debrand *et al.*¹² ventilated the lungs initially via a tracheal tube proximal to the stenosis. A second tube was passed through the distal nonstenotic trachea after the trachea was incised. The stenotic segment was excised, and a tracheal tube was passed retrogradely through the open proximal trachea and out through the mouth. Ventilation was continued through this tube whilst the tracheal repair was completed. Campbell and Lilly,¹⁰ in a report of the successful repair of a complete tracheal stenosis with associated right lung agenesis, describe stepwise advancement of the tracheal tube by opening four tracheal rings at a time and wedging the tracheal tube into the intact distal trachea. This was repeated until the entire anterior trachea had been split to the midportion of the left mainstem bronchus. Weber *et al.*¹³ describe intubation of the right and left main bronchi and ventilation of each lung individually during repair of a tracheal stenosis involving the carina. Ventilation was continued subsequently via a tracheal tube above the anastomosis.

HFJV has been found to be satisfactory for tracheal resection in adults¹⁴ and children.¹⁵ Schur *et al.*¹⁶ passed two catheters via the tracheal tube into the two main bronchi and ventilated the lungs at a rate of 80 breaths/minute. Positive pressure ventilation was reinstated after resection

and formation of the anastomosis. The advantages claimed over standard positive pressure ventilation include favourable haemodynamic effects, a still surgical field, less barotrauma, the effective clearance of the distal airways of blood and debris by the continuous flow of gas in a proximal direction, and better gas exchange. Minato *et al.*¹⁷ reported a case in which a tracheal bronchus was present proximal to a stenotic segment extending to the carina. The left lung was ventilated using HFJV whilst the tracheal bronchus was ventilated conventionally via a nasotracheal tube.

Both of the cases which we have described were managed using CPB. The advantages of CPB are that it permits excellent surgical access, avoids technical difficulties such as having more than one tracheal tube close to the operating site and enables simultaneous correction of cardiac defects. It allows enough time for an unhurried operation, ensures good oxygenation and gas exchange¹¹ and is the favoured technique at our hospital. The anastomosis should be tested for evidence of an airleak with an inflation pressure of 4 kPa (40 cm H₂O) before discontinuation of CPB.

Postoperative management

The trachea should be extubated as soon as possible because of the risk of suture line separation and air leak with positive pressure ventilation,¹⁸ and the risk of recurrent stenosis above the repair with prolonged tracheal intubation. The distal tip of the tracheal tube must be positioned correctly while the trachea remains intubated. The tip must be proximal to the anastomosis if tracheal resection has been performed. The nasotracheal tube may be required to act as a stent after tracheoplasty while the graft becomes incorporated into the tracheal wall. The neck may be held flexed for a number of days postoperatively by suturing the chin to the anterior chest wall, in order to minimise anastomotic tension. Humidification of inspired gases should be continued into the postoperative period to prevent thick secretions blocking the major airways. Occlusion of the tracheal lumen with a mucus plug in Case 2 resulted in a life-threatening deterioration in the patient's condition and was relieved only by emergency bronchoscopy. Tracheomalacia due to an inadequately supported tracheal wall may follow extubation. This was managed successfully in Case 2 with a nasopharyngeal prong to provide CPAP.

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Acute mercury poisoning

G. A. McLAUCHLAN

Summary

A case of deliberate self poisoning with approximately 7 g mercuric chloride (the lethal dose is about 1 g) is described. The patient died after 6 days despite the use of chelating agents, plasma exchange and intensive supportive therapy. The management of acute mercury toxicity is discussed.

Key words

Poisoning; mercury, mercury chloride.

Poisoning with mercury is rare and few units have experience of its management. A case is reported which, although fatal, provided important lessons relevant to the treatment of future patients.

Case history

A 23-year-old female postgraduate chemistry student presented to casualty 3 hours after taking 11 vitamin capsules that had been filled with approximately 7 g mercuric chloride. On arrival she complained of severe abdominal pain and was very distressed. Advice was sought from Guy's Hospital poisons unit immediately and throughout her admission.

Gastric lavage produced blood-stained fluid only. Four egg whites and 10 g charcoal were left in the stomach. Two litres saline 0.9% were given intravenously over the next hour.

Her condition deteriorated rapidly; respiration became more laboured, she vomited and became hypotensive. She tolerated tracheal intubation without sedation and her respiratory pattern and conscious level subsequently improved. Suction through the tracheal tube produced frothy fluid stained with blood and charcoal. She also began to bleed *per rectum*.

The first dose of intramuscular dimercaprol was administered approximately 4 hours after ingestion of poison. Some delay had occurred as the drug had to be brought from a neighbouring hospital. Intravenous diamorphine 3.5 mg was given for pain relief. The patient was transferred to the intensive care unit where she was sedated with morphine and midazolam, and intermittent positive pressure ventilation was instituted.

Bladder catheterisation produced 400 ml of urine but after this she was anuric. An arterial line was inserted in the right femoral artery (no other arteries were easily palpable) and a pulmonary artery catheter was inserted via the right subclavian vein. Initial blood gases showed a marked acidosis (pH 6.7, P_{O_2} 48.3 kPa, P_{CO_2} 7.0 kPa, base deficit 14.1 mmol/litre). The pulmonary artery wedge pressure was -1 mmHg. The cardiac output remained low (2.6 litres/minute) despite correction of the acidosis with bicarbonate and fluid replacement to bring the wedge pressure up to 10 mmHg. She required increasing cardiovascular support with dobutamine, dopamine and noradrenaline over the next few days. The systolic blood pressure was rarely above 80 mmHg despite these measures.

Chelator therapy was continued, dimercaprol being replaced by dimercaptopropane-1-sulphate (Dimival), an oral substitute, when this became available.

She received a 2.5-litre plasma exchange 20 hours after admission and continuous haemodialysis was started. She continued with this regimen daily; haemofiltration was substituted for haemodialysis on the fourth day. Daily samples were obtained for measurement of blood mercury concentration. Samples of dialysate fluid, filtrate fluid and exchanged plasma were also analysed (Table 1; Fig. 1).

Serial chest radiographs showed the progressive picture of adult respiratory distress syndrome and she became increasingly hypoxic. On the sixth day, she suffered a cardiorespiratory arrest from which it was not possible to resuscitate her.

Postmortem examination showed pulmonary changes consistent with severe adult respiratory distress syndrome and the cause of death was given as cardiorespiratory failure secondary to acute mercury poisoning. Mercury

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Table 1. Mercury concentrations in fluids and total removed daily by each method.¹⁷

1. <i>Haemofiltration fluid</i>	Concentration = 214 µg/litre
Amount removed in 24 hours = filtrate concentration × volume/hour × duration of filtration = 214 × 0.5 × 18 = 1926 µg.	
Clearance = Waste conc. × flow rate/plasma conc.	
= (214/1000) × (500/60)/5 = 0.35 ml/minute	
2. <i>Haemodialysis fluid</i>	Concentration = 9 µg/litre
Amount removed per 24 hours = dialysate concentration × volume/hour × duration of dialysis = 9 × 0.5 × 18 = 81 µg.	
Clearance = waste conc. × flow rate/plasma conc. = (9/1000) × (500/60)/6 = 0.0125 ml/minute.	
3. <i>Plasma exchange fluid</i>	Concentration = 1525 µg/litre
Amount removed per 24 hours = 1525 × 2.5 = 3812.5 µg.	
Clearance = waste conc. × flow/plasma conc. = (1525/1000) × (1000/60)/6 = 4.24 ml/minute.	

concentrations in various tissue samples obtained at postmortem are shown in Table 2.

Discussion

Mercury is a relatively freely available toxin and poisoning has occurred by most routes (from oral to intra-arterial) with varying outcome,¹⁻⁵ depending on the route, form and quantity taken.

Elemental mercury is poorly absorbed orally and as much as 204 g have been taken with little ill effect;¹ in contrast, inorganic salts of mercury can be fatal when less than 1 g is taken although only 15% is absorbed. The concentrations in our patient were the second highest ever recorded at Guy's poisons unit.

The initial effect of oral mercury is from proteolysis of the mucous membranes, which results in severe oral and abdominal pain, with ulceration of the affected surfaces. Gut resection was necessary to stop haemorrhage in severe cases; in less severe cases, hypovolaemic shock ensues. Our patient had ulcers at impact sites at postmortem but was spared the oral problems because mercury was ingested in an encapsulated form. She required fluid replacement out of proportion to the observed losses, presumably as a result of intra-abdominal fluid shifts and from 'leaky' capillaries. In future cases we would give more fluid from the outset.

Inorganic salts of mercury are absorbed quickly (the majority within the first 15 minutes) and are distributed

Table 2. Postmortem mercury concentrations (mg/litre) in tissues.

Brain	0.39
Colon	17.9
Heart	2.0
Liver	9.3
Lung	6.5
Kidney	20.8
Gastric contents	12.7

initially between red blood cells and plasma in roughly equal proportions (organic salts are concentrated within the erythrocytes). Systemic absorption affects all tissues at a cellular level. Mercury is known to bind to sulphhydryl groups, producing damage to cellular proteins and enzymes that contain these groups. It is not known whether this causes the widespread cellular damage that occurs in mercury poisoning. The organ affected first (the critical organ) depends on the form of the mercury; inorganic salts affect the kidney initially, and produce few central nervous system effects.

Treatment is both general and specific. Intensive multi-system support is required and attempts are made to reduce absorption, limit distribution and increase excretion. Whole blood mercury concentration is the best available assay and provides a better guide to total body load in acute poisoning than urine or hair concentrations.

Absorption

Recommended emergency treatment is gastric lavage followed by instillation of protein (egg white or milk) and charcoal to bind and absorb any mercury that remains. One risk of lavage in a sick patient is pulmonary aspiration. This occurred in our patient and probably contributed to the rapid deterioration; mercury is not noted for producing lung involvement unless aspirated.^{3,5} Elective tracheal intubation would have given protection and should be considered when any irritant substance is ingested, especially when the conscious level is altered.

Another point of interest in our case was the high postmortem mercury concentrations in the stomach. Some of this would be due to desquamation but repeated lavage or protein instillation might have reduced any reabsorption that may have occurred.

Distribution

The aim of using chelating agents is to bind the poison in the blood stream in a soluble form, thus speeding excretion and delaying further distribution. The chelates formed are unstable. Consequently repeated doses are required.

Various agents were used in the past, including acetyl cysteine, dimercaprol (British Anti-Lewisite), d-penicillamine and Dimival (dimercaptopropane-1-sulphate).⁷⁻¹¹ Dimercaprol is the most widely used agent for mercury poisoning but has many dose-related side effects that include nausea, fever, hypertension, tachycardia, and abdominal pain.¹² It must be given intramuscularly at 4-hourly intervals to maintain adequate blood concentrations. There is also a theoretical risk of increasing brain concentrations of mercury.¹⁴ Dimival has fewer side effects and can be given orally, but is not widely available. A chelating agent

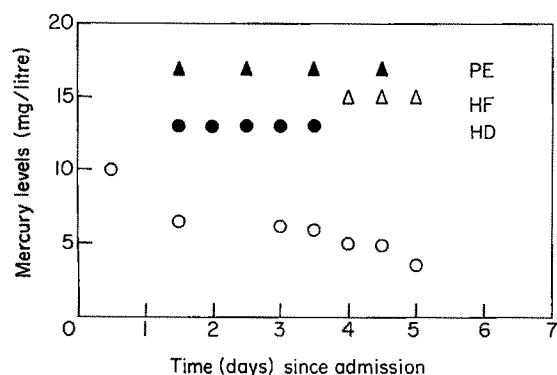


Fig. 1. Blood mercury concentrations (○) and their relationship to various interventions. PE, plasma exchange; HD, haemodialysis; HF, haemofiltration.

must be given urgently if renal protection is to be achieved. We encountered a short delay in obtaining supplies of dimercaprol; delays might be longer in a more peripheral hospital. Acetyl cysteine, which is readily available, may be a useful agent in the short term.

Excretion

Mercury chloride is excreted in the faeces and in the urine. Renal failure is likely if significant poisoning has occurred and alternative routes of excretion must be used. Different forms of dialysis have been tried (haemodialysis, peritoneal dialysis, haemofiltration and haemodialysis with acetyl cysteine infusions), all with limited success in reducing the body load. The most effective therapy, in terms of hourly removal of mercury (Table 1) is plasma exchange (in the case of organic salts, with heavier erythrocyte binding, whole blood exchange might be tried). This may be repeated frequently to increase clearance. Dialysis or haemofiltration may be required also to replace renal function.

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CASE REPORT

Pulmonary air embolism during laparoscopic laser cholecystectomy

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Summary

We describe a case of pulmonary air embolism that occurred during laparoscopic cholecystectomy using the neodymium:yttrium-aluminium-garnet laser.

Key words

Surgery; laparoscopic cholecystectomy.

Equipment; laser.

Complications; air embolism.

Cholecystectomy is now performed laparoscopically using the neodymium:yttrium-aluminium-garnet (Nd:Yag) laser. Several hundred cases have been performed in the United States^{1,2} and there is now an explosion of interest in this country. There are many advantages with this type of surgery compared with open cholecystectomy. Precise surgery may be performed through small incisions sufficient for the laparoscopic instruments. There is reduced operative blood loss and oedema, with sterilisation of the impact site. Postoperative pain and ileus are minimal, which allows patients to be discharged one or two days after surgery. There is unquestionable benefit to the patient and significant cost saving.³

The Nd:Yag laser is used with an artificial sapphire scalpel, the contact probe for surgery. The sapphire tip is protected from laser-induced thermal damage by the continuous flow of air, carbon dioxide or nitrogen transmitted via a sheathed fibre. The main complication of laparoscopic laser cholecystectomy is haemorrhage, which is difficult to control because the operative field is obscured; laparotomy may be necessary. We describe another, potentially fatal complication, that of intra-operative air embolism.

Case history

A 28-year-old, 52-kg woman was admitted to hospital for elective laparoscopic cholecystectomy. She had an 18-month history of intermittent right upper quadrant pain and ultrasonography demonstrated a thick-walled gall

bladder that contained a single stone. She had undergone two Caesarean sections 4 and 6 years previously. She smoked 15 cigarettes a day but otherwise no significant features of the history were noted. Clinical examination revealed no abnormalities. Full blood count, coagulation screen, liver function tests, serum urea and electrolytes were within normal limits. Blood group was determined and serum saved.

Premedication consisted of pethidine 100 mg and promethazine 25 mg administered intramuscularly 1 hour before anaesthesia. Anaesthesia was induced intravenously with thiopentone 350 mg and the trachea intubated with an 8-mm oral tube after atracurium 30 mg had taken effect. An intravenous infusion of compound sodium lactate solution was started, a nasogastric tube passed and a urinary catheter inserted. Anaesthesia was maintained with enflurane and nitrous oxide in 40% oxygen, and muscular relaxation with atracurium infused at 0.5 mg/kg/hour. Droperidol 2.5 mg and a total of 450 µg fentanyl in increments were given during the procedure.

Monitoring consisted of continuous electrocardiography, pulse oximetry, end-tidal capnography, inspired oxygen fraction, ventilator inflation pressure, core and peripheral temperature and urine output. A radial artery line was inserted for continuous arterial pressure monitoring and arterial blood gas estimation, since from previous experience it was expected that large volumes of carbon dioxide would be used to sustain the pneumoperitoneum required for this type of surgery.²

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Anaesthesia was uneventful until 55 minutes after the start of surgery. Systolic arterial blood pressure had been in the range 105–135 mmHg, pulse rate 70–80 beats/minute, end-tidal carbon dioxide 4–4.7 kPa and oxygen saturation 98%; arterial blood gases taken at 45 minutes gave a pH 7.42, P_{CO_2} 3.9 kPa, P_{O_2} 19.5 kPa and oxygen saturation 99%.

Suddenly, during the laser dissection of the gall bladder from the liver bed and shortly after the liver had been inadvertently pierced with the sapphire tip, there was a dramatic decrease in the end-tidal carbon dioxide to 1.5 kPa. The lungs were manually inflated with 100% oxygen once ventilator disconnection was excluded. Surgery was immediately halted and the abdomen emptied of carbon dioxide. Chest movements were symmetrical and auscultation of the lungs revealed no abnormality. The oxygen saturation gradually decreased to 65% and the patient became cyanosed. There had been no change in ventilator inflation pressure, and the pulse rate and arterial blood pressure were virtually unchanged. Pulmonary embolism was suspected. The end-tidal carbon dioxide had returned to 5 kPa within 5 minutes and the oxygen saturation to 95%. The inspired oxygen fraction was reduced to 0.5 and arterial blood gases taken at this time gave the following results: pH 7.34, P_{CO_2} 5.5 kPa, P_{O_2} 11.5 kPa and oxygen saturation 96%, which indicated a shunt. The ECG remained regular throughout the episode, but three ventricular ectopic beats were seen a few minutes afterwards.

The pneumoperitoneum was re-instated and surgery proceeded with the successful removal of the gall bladder via the laparoscope 45 minutes later. Reversal of neuromuscular blockade was achieved after skin closure, with neostigmine 2.5 mg and glycopyrronium 0.5 mg. Intra-operative fluid replacement was 2 litres of crystalloid. The nasogastric tube and urinary catheter were removed at the end of surgery and the patient tolerated oral fluids 6 hours later. Her postoperative recovery was complicated by the development of a mild, generalised wheeze on the first postoperative day. She then gave a history of having been similarly wheezy after her two previous general anaesthetics. There was no fever or calf tenderness and chest X ray was normal. She received three doses of paracetamol 1 g for analgesia before she was discharged on the second postoperative day.

Discussion

The features of this episode were a sudden decrease in end-tidal carbon dioxide, with oxygen desaturation and minimal cardiovascular changes in a paralysed patient undergoing laparoscopic surgery. Recovery occurred gradually over 5 minutes with the administration of 100% oxygen. These features are consistent with a small pulmonary venous air embolus. Pulmonary venous embolus was thought to be unlikely in a patient without significant risk factors for venous stasis.

It is relevant that this episode occurred shortly after the liver had been inadvertently pierced by the sapphire tip, with the laser inactive. The Nd:Yag laser was cooled with air flowing at 0.5 litres/minute from ports proximal to the tip, but at the time of the episode this was not known to the

anaesthetic team and so the aetiology of the pulmonary embolus was not immediately ascertained. For some 15 seconds these ports must have been buried in the liver and sufficient air therefore would have entered the hepatic venous system to have caused an air lock in the pulmonary circulation, failure of pulmonary gas exchange and the sudden changes seen in capnography and pulse oximetry. Fortunately there was no cardiovascular collapse, presumably because the laser tip was promptly withdrawn from the liver and the volume of air injected was relatively small. Capnography has been demonstrated as a reliable, sensitive, early method of detection of pulmonary air embolism.^{4,5}

Fatal gas embolism occurring during Nd:Yag laser endometrial ablation and during laser-induced hyperthermia for the treatment of hepatic metastases, has been described.^{6–8} These cases involved sapphire tips cooled with air or nitrogen, but these have now been abandoned in endometrial ablation in favour of bare quartz fibres which need no cooling. However, the bare fibres are unsuitable for the precise requirements of gall bladder surgery. The sapphire contact probes focus the laser beam over a short distance concentrating the energy so that cutting predominates over coagulation.

Air embolism presents a grave risk to the patient. It would be appropriate to use the Nd:Yag laser cooled with a more soluble gas, such as carbon dioxide, during surgery in highly vascular areas. Minimum gas flows should be used. A fluid-cooling medium, such as normal saline would avoid this risk, but is inappropriate because the sapphire tip is damaged by too rapid cooling. This case emphasises the importance of vigilant intra-operative monitoring of both the patient and of the surgical technique, when a gas-cooled laser is used.

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CASE REPORT

Williams syndrome: masseter spasm during anaesthesia

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Summary

A 4-year-old boy with Williams syndrome developed masseter spasm after halothane and suxamethonium. He did not develop malignant hyperthermia; the surgery was accomplished with a nontriggering anaesthetic and no further problems.

Keywords

*Complications; Williams syndrome.
Masseter spasm.*

Williams syndrome^{1,2} is a very rare disease associated with an elfin face, mental retardation and cardiovascular anomalies. The latter can take the form of supravulvar aortic stenosis and/or pulmonary supravulvar stenosis. All patients have the same characteristic facial appearance, partly determined by the mandibular hypoplasia. There are various dental anomalies including hypoplastic teeth. Idiopathic hypercalcaemia in infancy is thought to be associated with the syndrome. The aortic stenotic lesions seen in idiopathic hypercalcaemia and in experimental vitamin D intoxication are similar and it has been suggested that there might be a metabolic disorder *in utero*.²

Case history

A 4-year-old Caucasian boy who weighed 15 kg with Williams syndrome was presented for correction of left strabismus. His exercise tolerance seemed to be normal and he had had an uneventful general anaesthetic for bilateral herniorrhaphy. Tracheal intubation was considered potentially difficult because he had protruding top teeth and a relatively hypoplastic mandible. He was premedicated with diazepam 5 mg by mouth since he was known to be of a difficult temperament. Sodium citrate 7.5 ml was given before anaesthesia which was easily induced with halothane and nitrous oxide in oxygen. The trachea was intubated with a 4-mm oral Rae tracheal tube but he was subsequently extubated because of an excessive leak around the tracheal tube. He developed intractable laryngospasm during laryngoscopy for re-intubation that required a dose of muscle relaxant. Suxamethonium 10 mg was given but seemed to be ineffective and a further 5 mg was given.

Masseter spasm was evident after the second dose and consequently the halothane was discontinued. The masseter spasm resolved spontaneously over a period of about a minute, and vecuronium 1 mg was given to facilitate intubation. The 4.5-mm tracheal tube was a tight fit and he was extubated again and re-intubated with the original tube. A throat pack was inserted. Anaesthesia was maintained with nitrous oxide in oxygen and 5 µg fentanyl. He required no further muscle relaxant. No other features of malignant hyperthermia were seen.

The patient showed no signs of awareness during the procedure and was extubated awake at the conclusion of the surgery. There were no postoperative problems. Biochemistry performed a week after operation was within normal limits. The creatine kinase was 122 IU/litres. A muscle biopsy was not performed nor was the urine myoglobin measured.

Discussion

There is only one report³ in the anaesthetic literature on Williams syndrome. Laryngoscopy was not difficult in our case although intubation problems should be considered because of the dysmorphic features already mentioned. Contrary to the report of van Beuren *et al.*¹ this patient had a difficult temperament. Hypotonia may be present in Williams syndrome,² and this disorder of muscle tone should warn of a possible interaction with anaesthetic agents. There are, however, no data that suggest an increased incidence of masseter spasm or malignant hyperthermia in these patients. The incidence of masseter spasm varies depending on the anaesthetic technique. It is

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Fig. 1. The patient with Williams syndrome. Note the epicanthic folds, depressed nasal bridge, prominent lips with open mouth, mandibular hypoplasia and the low set ears.

higher in patients induced with halothane and suxamethonium than those induced with thiopentone and suxamethonium.⁴⁻⁷ An incidence of about 1% in children anaesthetised with halothane and suxamethonium has been reported.^{5,7}

It is well recognised that the incidence of masseter spasm is greater in patients undergoing strabismus surgery. Carroll⁵ found a fourfold difference when comparing the incidence in strabismus patients with patients without strabismus.

Christian *et al.*⁸ found a much lower incidence of masseter spasm in children, approximately 1:100 000, but they assumed that they were reporting a different condition or reporting a more severe degree of the same condition. There is some debate in the literature whether masseter spasm should still be regarded as an early sign of malignant hyperthermia.

Van der Spek *et al.*⁹ measured mouth opening after induction of muscle relaxation with suxamethonium, pancuronium and vecuronium in children and found there was a highly significant reduction in mouth opening and increase in jaw stiffness with suxamethonium ($p < 0.0001$). Patients who received pancuronium or vecuronium did not show this effect. It would appear that an increase in masseter muscle tone with suxamethonium is normal, and an exaggerated response may clinically be taken to be masseter spasm.

This child exhibited excessive masseter tone after the use of halothane and suxamethonium; this may have been an exaggerated normal response, but in the presence of other

congenital abnormalities of the musculoskeletal system, the danger of malignant hyperthermia was considered a real possibility and appropriate action taken.

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Epidural blood patch for treatment of subarachnoid fistula in children

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Summary

A subarachnoid-cutaneous fistula developed in two children after the placement of a catheter in the subarachnoid space for drainage of cerebrospinal fluid. The management of this iatrogenic complication with epidural blood patch is described.

Key words

Anaesthetic technique, regional; epidural, blood patch. Complications; fistula.

The drainage of cerebrospinal fluid (CSF), by placement of a catheter in the subarachnoid space is often performed to allow surgical exposure and to prevent leakage of CSF from dural wounds.¹⁻⁴ A rare but a distressing complication of this technique is the development of subarachnoid cutaneous fistula. Epidural blood patch has been successfully used to treat CSF fistula in adult patients,⁵⁻⁶ and we report its use in the management and treatment of two children with this type of fistula.

Case histories

Patient 1

A 6-year-old boy was struck near the left orbit with a golf club. There was no history of loss of consciousness or any other neurological symptoms except for several bouts of vomiting in the emergency room. X ray of the skull showed a nondisplaced left blow-out fracture of the orbit and ethmoid bone associated with pneumocephalus along the left hemisphere and suprasellar cistern.

The patient underwent emergency exploration and repair of the laceration on the eyelid. A CSF leak was observed, during exploration, from the undisplaced fracture in the ethmoid. Remaining surgery was uneventful and the patient was confined to bed after operation, with the head of the bed elevated at 45–60° for 36 hours. CSF continued to drain from the nose and a 16-gauge plastic catheter was placed via a 14-gauge needle in the interspace between the third and fourth lumbar vertebrae to drain CSF. The CSF leak from the nose ceased 6 days after the placement of the

catheter, which was removed 3 days later, but a clear fluid (glucose positive) continued to drain from the catheter placement. The CSF leak from the lumbar region persisted for 2 days despite strict bedrest. The child's neurological examination remained normal throughout this period, and he denied having any headaches. The drainage was estimated at approximately 100 ml per 8 hours by weighing wet dressings.

The patient was referred to the pain clinic for epidural blood patch on the third day after removal of the catheter and was sedated with 400 mg of rectal 2.5% methohexitone, and 30 µg of intravenous fentanyl. A 2.5 inch 20-gauge Tuohy needle was placed in the L₄₋₅ interspace, and the epidural space identified by the loss of resistance technique. Seven ml of autologous blood were injected into the epidural space under aseptic conditions. The patient was confined to bed for the next 24 hours, during which CSF continued to leak from the lumbar region, but in smaller amounts as judged by fewer changes of dressings. The epidural blood patch was repeated the next day, after sedation with fentanyl 60 µg, midazolam 1.5 mg and ketamine 45 mg titrated intravenously over 5–10 minutes. The L₂₋₃ interspace was used on this occasion and 8.5 ml of autologous blood injected into the epidural space. A figure-of-eight suture was placed around the opening of the fistula and the patient confined to bed for another 24 hours. He was gradually allowed to walk during the next 48 hours and the suture was removed at the end of this period. There was no evidence of CSF leak from nose or lumbar region, and he was discharged home 2 days after the second epidural blood patch.

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Patient 2

A 6-year-old white girl presented with a 3-week history of proptosis. Computerised tomography of her head revealed a large ethmoidal mass with extension into the maxillary and sphenoidal sinuses. Biopsies of the mass were consistent with fibrous dysplasia and the patient underwent a 22-hour resection and reconstruction procedure of the orbit under general anaesthesia. A 16-gauge plastic catheter was placed before the start of surgery in the subarachnoid space at the L₃₋₄ interspace, passed through a 14-gauge needle to facilitate exposure of the surgical field and prevent post-operative CSF leak. There was no evidence of CSF leak from the surgical wound site and the catheter was removed 72 hours after the initial placement. Clear glucose-positive fluid drained from the site of the catheter placement a day later. The patient was confined to bed for the next 4 days, during which subcutaneous infiltration around the fistula with saline was attempted, on two occasions without success. On the 5th day she was referred to the pain clinic for an epidural blood patch, and was sedated with intravenous fentanyl 25 µg and the epidural space was located using the loss-of-resistance technique with an 18-gauge Hustead needle at the L₄₋₅ interspace. Eight ml of autologous blood was injected into the epidural space with cessation of CSF leak, and a figure-of-eight suture was placed around the opening of the fistula. The patient was confined to bed for 48 hours at which time the suture was removed and there was no further CSF leak.

Discussion

Subarachnoid-cutaneous fistula following lumbar puncture for surgical or therapeutic procedures is a rare but serious complication. It is quite distressing to the patient and also acts as a portal of entry for bacterial infection.⁴ The incidence of fistula formation depends on the duration of subarachnoid drainage and the diameter of the catheter. Conservative measures or surgical correction^{7,8} are time consuming and expensive, and the use of an epidural blood patch for treating persistent CSF leak into the epidural space after dural puncture is well documented.⁹⁻¹¹ The technique, since its description by Gormley in 1960, has been a very effective method of treating postspinal puncture headaches,^{12,13} although complications such as headaches, back stiffness, radicular nerve discomfort^{13,15} and subdural hematoma¹⁶ are reported.

The efficacy of epidural blood patch for treatment of CSF fistula in an adult was reported previously.^{5,14} The volume of blood recommended in adults for treatment of postspinal headache varies from 10 to 20 ml^{10,13} although as little as 3–5 ml⁹ is reported to be effective. Alacreu *et al.*⁶ reported a case in which 22 ml of autologous blood was injected into the caudal space of a 12-year-old patient to treat CSF fistula.

It is not necessary to inject the blood as close as possible to the dural puncture or CSF fistula,⁶ but it is suggested that it is desirable to place the blood caudal to the fistula or the hole in the dura, since Szeinfeld *et al.*¹⁷ demonstrated that the blood injected into the epidural space has a greater tendency for cephalad spread. We placed blood caudal to

the fistula in both our cases, which resulted in complete cessation of CSF leak in the second case, but only partial closure of the fistula in the first. The blood was placed rostral to the fistula for the second blood patch (first case) which resulted in complete cessation of the CSF leak.

It was shown by Rosenberg and Heavner¹¹ that some of the blood injected into the epidural space adheres to the dural defect and forms a clot, thereby sealing the puncture site. An endothelial tube probably extends from the dura to the skin in the case of a CSF fistula. The mechanism by which an epidural blood patch interferes with a fistulous tract is unknown, but the pressure generated by the blood clot may compress the fistula directly, thereby obliterating it. The treatment of this iatrogenic complication with an epidural blood patch is a useful, economical and effective alternative to surgical or conservative measures.

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CASE REPORT

Acute airway obstruction, hypertension and kyphoscoliosis

P. M. C. WRIGHT AND J. P. ALEXANDER

Summary

An elderly woman developed acute respiratory obstruction after choking on a bolus of food. On rigid bronchoscopy no foreign body, or anatomical obstruction was seen, but airway obstruction recurred during emergence from general anaesthesia, and was thought to be functional in nature. A recurring airway obstruction followed, relieved by induction of anaesthesia and by sedation. This was subsequently demonstrated to be caused by a tortuous aorta which impinged intermittently on the anterior tracheal wall, as a result of kyphoscoliosis of the thoracic spine. We attribute the relief of airway obstruction by general anaesthesia to a reduction in arterial blood pressure.

Key words

Airway; obstruction, trachea, thoracic aorta.

Complications; kyphoscoliosis.

Upper airway obstruction from vascular pressure on the tracheobronchial tree occurs most frequently in the neonatal population,¹ and it is most unusual for a case of this type to present in adulthood. The following case illustrates that vascular pressure can cause tracheal collapse even in the elderly and may cause difficulties in diagnosis.

Case history

An 86-year-old Caucasian female presented in an Accident and Emergency department complaining of difficulty in breathing after choking on a food bolus about 1 hour before. There had been a bout of coughing that lasted about 30 minutes after the choking episode, after which the symptoms had abated.

The medical history included an uncomplicated myocardial infarction that occurred 3 years before, at the time of which hypertension was diagnosed and treated with triamterene 50 mg and hydrochlorothiazide 25 mg daily.

The patient was a frail, elderly, Caucasian female of slight build; her pulse was 88, the arterial blood pressure 150/70 mmHg, and there was a considerable kyphosis. She appeared comfortable initially and her colour was normal, although there was some stridor. During the examination, however, she deteriorated and experienced difficulty in breathing accompanied by loud stridor and cyanosis. A presumptive diagnosis of partial airway obstruction from a foreign body was made. Reliable venous access was secured and oxygen administered from an anaesthetic system while

the patient was transferred directly to the operating theatre for a rigid bronchoscopy.

Anaesthesia was induced with halothane in oxygen from a Magill system, and the stridor and respiratory obstruction improved. Rigid bronchoscopy and oesophagoscopy revealed only some reddening of the trachea, and no foreign body was seen. After the procedure, oxygen was administered by facemask while recovery from anaesthesia took place. Her respirations continued to obstruct intermittently before full recovery, which caused distress and stridor. Laryngoscopy was performed and the trachea intubated with a cuffed oral tracheal tube, and she was subsequently admitted to the Intensive Care Unit (ICU) for further management. She was noted to be alert and comfortable on admission to the ICU, despite the presence of a tracheal tube and was able to communicate by hand signs. Pulse was 110 and arterial blood pressure 135/80 mmHg. Arterial blood gas analysis showed a P_{aO_2} of 10.73 kPa, P_{aCO_2} of 5.13 kPa and a pH of 7.42, while breathing humidified 30% oxygen via a T-piece. A diagnosis of functional stridor was made in view of the normal bronchoscopic findings.²

The next day she was alert and comfortable, her colour was normal and chest examination revealed only the kyphosis; she was reassured and the tracheal tube was removed after suction to the oropharynx. No further symptoms appeared until 2.5 hours later when she developed respiratory distress with loud stridor. Inhalation induction of anaesthesia was performed with oxygen and isoflurane,

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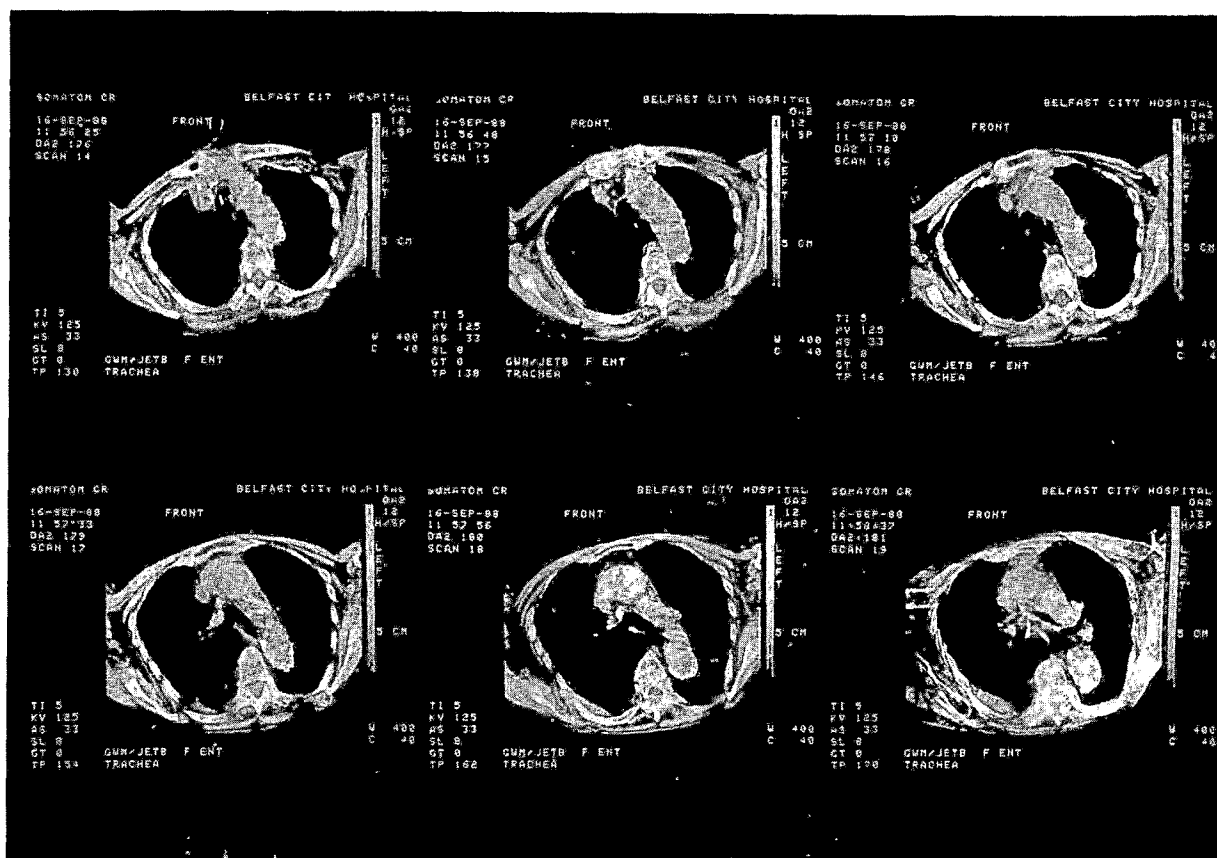


Fig. 1. Serial CT sections that demonstrate unfolding of the thoracic aorta which gave rise to airway obstruction.

and the respiratory obstruction disappeared. Laryngoscopy revealed a clear view of the larynx and trachea to the carina; no abnormalities were seen. The trachea was again intubated and the patient allowed to awaken.

The patient removed the tracheal tube herself 1.5 hours later, and she remained symptom free for a further 2 hours, at which point the symptoms recurred. An attempt was made to treat her with reassurance and small doses of sedation. Oxygen therapy was continued and midazolam 1 mg administered. This resulted in some sedation and an improvement in the stridor, with a clinical improvement in gas exchange. The improvement was short lived, after 30 minutes stridor recurred and the trachea was again intubated. She remained comfortable without sedation, and nutrition was provided with a liquid diet through a fine nasogastric tube.

Two days later a further attempt at extubation was made. Anaesthesia was induced with nitrous oxide and oxygen with isoflurane from a Magill system, but stridor returned immediately after the tracheal tube was removed. Laryngoscopy revealed tissue prolapsing into the tracheal lumen from the anterior, distal to the vocal cords. The trachea was reintubated and formal bronchoscopy arranged.

Rigid bronchoscopy the next day revealed contact ulceration below both arytenoids and on the anterior tracheal wall. Anterior midtracheal collapse was confirmed, a tracheostomy performed and in the course of this, a pulsatile vascular structure was noted impinging on the anterior tracheal wall. Arch aortography was arranged. This revealed unfolding of the aorta and showed that both the aorta and the innominate artery had a tortuous course.

Computer tomography of the chest was also performed, which confirmed the gross distortion of the thoracic cage due to kyphosis. The unfolded aorta is demonstrated lying immediately adjacent to the trachea (Fig. 1). The cause of her respiratory obstruction was collapse of the anterior tracheal wall as a result of pressure from the aorta resulting from kyphosis. Subsequently, in a further operation, a Montgomery T-tube was inserted to stent open the trachea. Her subsequent course was poor, marked by repeated chest infections, and she died 3 months after her initial presentation.

Discussion

Acute airway obstruction is a serious life-threatening condition in both adults and children, and anaesthetists may be involved in the management of this condition during diagnosis, emergency treatment and in intensive care. Diagnoses that must be considered in the adult include foreign bodies, laryngeal diseases including infection or malignancy, other respiratory tract infections or pressure from outside the respiratory tree, such as a goitre.

This case illustrates some interesting difficulties with diagnosis. Bronchoscopy failed to demonstrate any anatomical obstruction to the airways. Apparently normal findings supported a diagnosis of a functional or hysterical stridor, which was strengthened as a result of the disappearance of respiratory obstruction during the induction of anaesthesia and after sedation has been administered.³

Hysterical or functional stridor has been recognised more frequently in recent years.⁴ It is thought that a patient with functional stridor will not develop respiratory failure,

although intubation has been required to ensure the comfort of the patient in the short term.⁵

Deficiency of the anterior tracheal wall is a recognised complication of external pressure, most commonly as a result of thyroid goitre.⁶ The deficiency in the present case was from pressure from an unfolded aorta in combination with kyphoscoliosis and hypertension. We suggest that the reduction in symptoms that occurred with induction of general anaesthesia was a result of a decrease in arterial blood pressure and this also led to the erroneous diagnosis of hysterical stridor.

Fibreoptic laryngoscopy at the time of the symptoms has been recommended for confirmation of functional stridor⁷ and indeed, if used in this case, would have been more likely to demonstrate the collapsing anterior tracheal wall than rigid bronchoscopy under general anaesthesia. In view of the history of inhaled foreign body, however, rigid bronchoscopy was the necessary first investigation.

In elderly kyphotic patients presenting with the sudden onset of upper airway obstruction, the possibility of the aorta encroaching upon the tracheal wall should be con-

sidered. A reduction in arterial blood pressure may reduce symptoms and cause the disappearance of clinical signs.

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CASE REPORT

Ketamine, midazolam and vecuronium infusion

Anaesthesia for Down's syndrome and congenital heart disease

D. P. RILEY AND L. J. McBRIDE

Summary

A ketamine, midazolam and vecuronium infusion was used for total intravenous anaesthesia in a patient with Down's syndrome, a ventricular septal defect and pulmonary hypertension. A mixture of ketamine 200 mg, midazolam 5 mg and vecuronium 12 mg in 50 ml of normal saline was infused at 0.5 ml/kg/hour. This simple technique and ventilation with 100% oxygen maintained tissue oxygenation and cardiovascular stability.

Key words

Anaesthesia, intravenous; ketamine, midazolam, vecuronium. Anaesthetic techniques; continuous infusion. Congenital heart disease.

Patients with ventricular septal defects and pulmonary hypertension present special problems for the anaesthetist. The choice between general anaesthesia and local/regional analgesia, the use of nitrous oxide and volatile agents and the need to maintain systemic vascular resistance all demand careful consideration. We present a case of a patient with Down's syndrome and congenital cardiac disease in whom total intravenous anaesthesia was used, as described previously for use in military surgery.¹

Case history

A 42-year-old, 54-kg female with Down's syndrome, subaortic ventricular septal defect, patent ductus arteriosus and severe pulmonary hypertension presented for elective removal of a cataract and intra-ocular lens implant. It was considered necessary to replace the lens, despite her severe cardiac disease, since she enjoyed dancing and her reasonable lifestyle was progressively impaired by bilateral cataract disease.

On physical examination she had clubbing, central cyanosis, a right ventricular lift, increased pulmonary component of P₂, blood pressure of 120/80 mmHg and heart rate of 90 beats/minute. Her jugular venous pressure was not raised and her chest was clear to auscultation.

Her haemoglobin was 22.2 g/dlitre, platelets were 46 000, and she had a prothrombin ratio of 1.3 and aPTT 35. Echocardiography was reported as showing good left ventricular function and a hypertrophied, although not greatly dilated, right ventricle.

The patient was premedicated with intramuscular papa-

veretum 15 mg and hyoscine 0.3 mg one hour before surgery. After intravenous cannulation, a single dose of ceftriaxone 1 g was given for prophylaxis, and a left radial arterial line was inserted for direct blood pressure monitoring. Oxygen saturation by pulse oximetry on room air was 82–86%.

Anaesthesia, after pre-oxygenation, was induced with midazolam 1 mg, ketamine 20 mg, vecuronium 6 mg and the lungs were ventilated with 100% O₂. An infusion of ketamine 200 mg, midazolam 5 mg and vecuronium 12 mg in 50 ml of normal saline was started at 0.5 ml/kg/hour. The trachea was intubated easily and artificial ventilation performed with a Campbell's ventilator. Direct laryngoscopy promoted a self-limiting bradycardia to 50 beats/minute.

Oxygen saturation was maintained at 92–94% by ventilation with 100% O₂ throughout the procedure. Two episodes of bradycardia to 50 beats/minute (from 80 beats/minute) occurred with traction on the orbit by the surgeon, for which atropine 300 µg was given. The systolic blood pressure was maintained at 120–130 mmHg throughout the 2-hour procedure. Two doses of metaraminol 0.5 mg were given when the systolic blood pressure decreased to 110 mmHg.

The infusion was stopped 10 minutes before reversal of neuromuscular block by neostigmine 2.5 mg and atropine 1.2 mg given over 5 minutes, which avoided tachycardia.

There was slow recovery which allowed extubation of the trachea 30 minutes after the infusion was stopped. The patient was transferred to the Intensive Care Unit where she remained drowsy for 6 hours. Her blood pressure and

pulse rate were stable and oxygen saturation with 40% oxygen mask was 82–86%.

The patient was discharged home 48 hours after operation with an improvement in her vision to the degree of being able to count fingers.

Discussion

The principles of anaesthesia in patients with severe pulmonary hypertension and Eisenmenger's syndrome are to maintain tissue oxygenation in the face of altered cardiovascular dynamics. A right-to-left shunt diverts pulmonary blood flow from the pulmonary circulation to the systemic circulation with the result that any reduction in systemic vascular resistance (or increase in pulmonary vascular resistance) may lead to a reduction in pulmonary blood flow, increased venous admixture and reduction in P_{aO_2} . Maintenance of the systemic vascular resistance is necessary for tissue oxygen delivery.

The pathophysiology of the congenital cardiac lesions, the associated mental retardation from Down's syndrome, the specific type of surgery with the need for normal intra-ocular pressure and slow controlled emergence were important in the care of this patient. To satisfy these requirements it was decided to use an infusion of ketamine, midazolam and vecuronium to maintain cardiovascular stability as has been proposed for military surgery. The use of inhalational agents was avoided to keep systemic vascular resistance at a satisfactory level and the use of nitrous oxide avoided because it may have deleterious effects on pulmonary vasculature in patients with existing raised pulmonary vascular resistance.³ We used 100% oxygen since medical air was not available, although, had we the option, we would have considered the use of 50% oxygen. Local or regional techniques were avoided because of the mental retardation in this patient with Down's syndrome.

The surgeons reported operating conditions as excellent despite reports that ketamine increases intra-ocular pressure.⁴ The emergence was planned to be slow and the patient remained drowsy for some hours after operation. This slow emergence was considered an advantage to avoid major cardiovascular changes and coughing and straining, but it was possibly excessively long and in this particular patient's case we considered that the dose of midazolam in the infusion could have been reduced or, alternatively, the infusion stopped earlier. No emergence reactions were reported by the patient or nursing staff. The combination of midazolam with ketamine presumably prevents these reactions.^{5,6} Rapid response to the administration of intravenous midazolam and ketamine was seen as would be predicted with the right-to-left shunt.²

We present a case of Eisenmenger's syndrome with some unusual anaesthetic and operating requirements for which the use of total intravenous anaesthesia was very satisfactory for maintaining cardiovascular stability in this patient.

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CASE REPORT

Accidental injection of ether into the epidural space

A. MAPPES AND H. M. SCHAEER

Summary

Five ml diethyl ether was accidentally injected into the epidural space via an epidural catheter at the end of a Caesarean section. Immediately the patient felt a hot and burning pain in her legs that led to paraparesis, with a sensory level to T₈. The patient showed bilateral recovery of both motor and sensory functions 4 hours after the accident.

Key words

Anaesthetic techniques, regional; epidural. Complications; accident.

Bromage was one of the first to report cases of accidental injection into the epidural space. Recently the increased popularity of epidural analgesia has resulted in a higher incidence of reports of such complications, and we report the accidental injection of ether through an epidural catheter.

Case history

A 40-year-old woman who had three previous pregnancies presented in the 40th week for an elective Caesarean section because of Rhesus incompatibility of the fetus. The patient expressed a wish for epidural analgesia.

An epidural catheter was inserted 5 cm at the L_{2/3} interspace, on arrival in the operating room. A test dose of 3 ml bupivacaine 0.5%, without adrenaline was given, after which a total of 15 ml bupivacaine 0.5% was given. A sensory block to the level of T₂ was established at the beginning of the operation, tested by warm–cold discrimination with ether. The cardiovascular variables were stable, and the operation lasted 50 minutes without any complications; the anaesthetist gave no further medication.

Sensation at the end of the operation extended to a level of T₆ and the patient was able to move her toes. At that point, according to our usual practice, 2.5 mg preservative-free morphine diluted in NaCl 0.9% (a 5-ml solution) should have been injected through the epidural catheter. Unfortunately, the ether syringe was mistaken for the morphine syringe, which resulted in an injection of 5 ml of

ether (pH 7) into the epidural space. The anaesthetist realised the error immediately.

The patient complained 2 to 3 minutes after the injection about 'hot legs' and, seconds later, burning pain in both legs (especially in the calves), that spread up to the abdomen. Very soon paralysis developed with a sensory block to the level of T₈. The patient became very tired in the next 30 minutes, but stayed awake and orientated. Blood pressure, ECG and pulse oximetry showed no significant changes.

The patient no longer complained about tiredness after one hour and was again able to bend the knees slightly and move her toes. Slight pain, however, persisted in both legs, but after 3 hours she was able to bend both knees fully and the pain had ceased.

A neurological examination by the anaesthetist 4 hours after the accident showed no abnormalities, and full mobilisation was possible 9 hours after the incident without any problems. Two neurological examinations, 24 hours and 10 days later, showed no pathological signs, but the patient failed to attend for a further neurological check-up at one month.

A few minutes after the accidental injection we tried to aspirate the ether solution from the epidural space, with little success (0.5 ml). Then we made three attempts to rinse the epidural space with 10 ml NaCl 0.9% through the epidural catheter; we aspirated 2–3 ml of the injected solution each time. Finally, we injected 100 mg prednisolone, diluted with 10 ml NaCl 0.9%.

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The epidural catheter stayed in place for 6 hours and was then removed. There was no evidence of damage to the material of the catheter.

Discussion

Only very rarely has persistent neurological damage been documented after accidental epidural injection. The agents were: hypertonic sodium chloride,² colloidon,² and potassium chloride.³ There were, fortunately, no persisting neurological deficits in all other reported incidents: thiopentone (four), potassium chloride (three), ephedrine (three), diazepam (two), methohexitone and magnesium sulphate.^{1,4-11} The small number of publications may not represent the actual number of accidental injections into the epidural space, but almost every substance found in the surroundings of the patients could be accidentally administered into the epidural space.

We found no reports on inadvertent epidural ether injections in man in the international literature, although the routine use of ether through syringes or by sponges to assess the level of regional analgesia is widely used. Theoretically, this very lipophilic substance is potentially toxic to the nerve roots.

Our experience leads us to suggest that the manoeuvres that we performed, aspiration through the epidural catheter, carefully rinsing the epidural catheter with NaCl 0.9% (2-3 times 10 ml NaCl 0.9%) and repeated neurological examinations, may have helped to alleviate the situation. We do not know whether the steroid injection was of any benefit. We immediately informed the patient about the accident; later we could tell her that if ether, thiopentone, ephedrine, diazepam, methohexitone or magnesium-sulphate was accidentally given into the epidural space the prognosis is good.

The therapeutic injection of corticosteroids or hyaluronidase has been advocated in the literature, but their beneficial effect is not proven, and the ether test to verify the local anaesthesia level should in future only be performed with an ether-soaked sponge or with syringes that contain ether mixed with a coloured substance (e.g. patient blue V, colour index 42051), to enable immediate identification.

We also conclude that in general, all syringes should be clearly marked with different coloured labels which identify the contents.

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CASE REPORT

Anaesthesia for Proteus syndrome

J. H. PENNANT AND M. F. HARRIS

Summary

A 14-year-old boy with Proteus syndrome presented for orthopaedic surgery to his legs. No report in the literature exists on anaesthesia for this condition. Our patient posed airway problems that were managed by tracheal intubation under sedation using a fiberoptic bronchoscope. Anaesthesia was induced with thiopentone and maintained with nitrous oxide and isoflurane in oxygen. His lungs were ventilated mechanically throughout surgery, which was uneventful.

Key words

Complications; Proteus syndrome.

Proteus syndrome was first described by Wiedemann *et al.* in 1983.¹ It presents with a variety of abnormalities, but is essentially a congenital hamartomatous disorder. Features relevant to anaesthesia include scoliosis and cystic lung changes. We describe the management of a severe case and review the literature on this rare disease.

Case history

A 14-year-old boy who weighed 35 kg with an established diagnosis of Proteus syndrome presented for correction of a club foot deformity and femoral epiphysiodesis. He appeared to be normal at birth but gradually developed the characteristic abnormalities of Proteus syndrome.

At 2 years he underwent a craniotomy for removal of an astrocytoma, and a ventriculo-peritoneal shunt was inserted. There was no residual neurological deficit other than seizures which were well controlled. Hemihypertrophy of the right side of his body caused a 10-cm limb length discrepancy which resulted in difficulty in walking and a secondary club foot deformity of the left leg. Severe kyphoscoliosis had developed after increased growth of the right side. At 9 years of age a posterior thoracolumbar spinal fusion with Luque rod instrumentation was performed at another hospital, from which no anaesthetic records were available, but he developed a deep vein thrombosis of the right leg after operation. Surgery corrected his thoracic and lumbar curvatures, but scoliosis rapidly progressed in his cervical spine, and within 4 months he had a fixed torticollis with his head held in a flexed, laterally rotated position.

The present operation was intended to correct his club foot deformity to allow walking, and to ablate the growth epiphyses of his hypertrophied right leg so that natural growth of the left side would equalise limb length. Severe craniofacial deformities were obvious on admission (Fig. 1). There was gross bossing of the skull in the right frontal and parietal areas, and a retrobulbar dermoid had proptosed the right eye such that the lids would not close. He was of normal intelligence. The cervical spine was completely fixed in a flexed, laterally rotated position. His prominent carious teeth were haphazardly arranged and full mouth opening was limited to 0.75 cm. Examination of the cardiovascular and respiratory systems was unremarkable. His mother volunteered that partial airway obstruction occurred during sleep. Full blood count, serum electrolytes and liver function tests were within the normal range.

X rays of the cervical spine revealed severe deformities (Fig. 2). Many cervical vertebrae showed hemihypertrophy and osteopenia. Flexion and extension views failed to demonstrate any mobility.

Regional analgesia was refused by the patient, and in view of his posterior spinal fusion was contraindicated. We elected to use, under sedation, a fiberoptic bronchoscope to intubate the trachea. He was brought to the operating theatre after premedication with morphine and hyoscine. An ECG and pulse oximeter were used to monitor vital signs. Further sedation was given with increments of fentanyl to 0.1 mg and midazolam to 2.75 mg, and he was given a gargle of 3 ml viscous lignocaine 2%. A 6.0-mm cuffed tracheal tube was positioned within a No. 8 Berman

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Fig. 1. Proteus syndrome: Note the fixed torticollis, frontal bossing, proptosis of the right eye and poor dentition.

Intubating/Pharyngeal Airway (Bay Medical Inc., Clearwater, Florida) that had been coated with lignocaine ointment 5%.² The airway was gently advanced past the teeth until its tip lay in the posterior pharynx. A 4.5-mm diameter Olympus LF-1 fiberoptic bronchoscope was intro-

duced through the tracheal tube held within the Berman airway and passed into the trachea. The epiglottis was noted to be enlarged and difficult to circumvent.

Anaesthesia was induced with thiopentone 125 mg and maintained with 65% nitrous oxide and 1% isoflurane in oxygen after confirmation of the correct positioning. Ventilation was controlled throughout the 2-hour procedure, without need for muscle relaxants. Direct laryngoscopy during surgery did not allow us to see the epiglottis. Indeed, no structure distal to the uvula was visible. The tracheal tube was removed when he awoke at the end of the operation. The only problem after operation was intermittent upper airway obstruction which, according to his mother, was normal for him. Pulse oximetry and supplementary oxygen were employed in postoperative management as needed. A postoperative visit 4 days later found him asleep with a similar degree of obstruction.

Discussion

Wiedemann first described the Proteus syndrome in four boys.¹ Named after Proteus, the 'Old Man of the Sea' of Greek mythology who took on many forms to avoid capture, this condition also has variable presenting features. These include partial bilateral gigantism of the hands and feet (macroductyly), hemihypertrophy, often of one whole side of the body, pigmented naevi, thickened skin on the palms and soles ('moccasin feet'), mesodermal hamartomatous tumours, and macrocephaly with cranial hyperostoses.³ Other observations include cystic lung disease,¹ venous varicosities and anomalous teeth.^{4,5} Cardiac disease is not seen. Seizures may reflect cerebral involvement, although in our patient they were a result of neurosurgery. Intelligence is usually normal. Scoliosis is a common finding,^{4,6-8} and 20% of patients have an elongated neck^{4,7,8} secondary to vertebral enlargement.

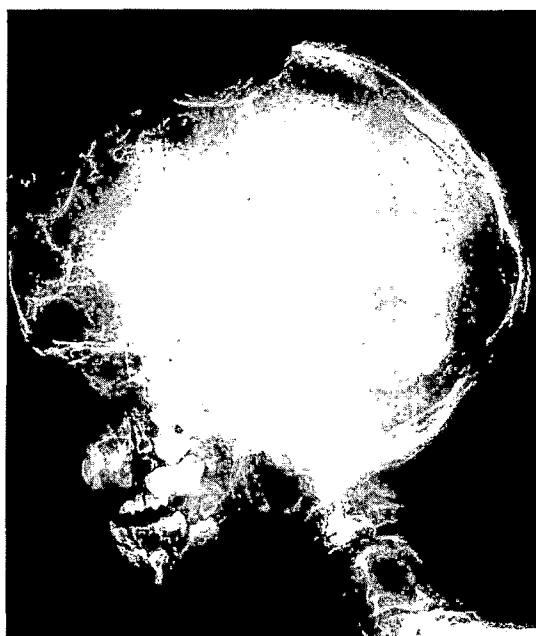


Fig. 2. Antero-posterior and lateral X rays of the skull and cervical spine taken with maximal mouth opening demonstrate marked frontal bossing and gross deformity of the spine. The upper end of a Luque rod is visible.

Hemivertebrae, dysplastic vertebrae, dystrophic intervertebral discs and spondylomegaly are often seen.^{8,9} Our patient displayed all the abnormalities described, including grossly enlarged cervical vertebrae. However, fixed torticollis has not been reported before in Proteus syndrome.

The disease appears soon after birth and progresses at a variable rate leading to gross deformity in later childhood. The prognosis is unknown, as is the potential for malignant change. The oldest reported patient is 24 years old, but one severe case lived only 16 months.⁶ Not all features manifest in all patients, and many earlier cases were probably misdiagnosed as neurofibromatosis,¹⁰ Bannayan-Zonana syndrome⁷ or Klippel-Trénaunay-Weber syndrome. Less than 40 cases have been described; the most famous was Joseph Merrick, the 'Elephant Man' presented by Sir Frederick Treves to the Royal Pathological Society of London in 1884. Merrick almost certainly had Proteus syndrome rather than neurofibromatosis.¹⁰ All cases appear to be sporadic mutations influencing the regulation of tissue growth factors.

No publications exist on anaesthesia for this condition so we do not know if tracheal intubation problems are common, but as there is a high incidence of cervical spine involvement, we anticipate them to be significant.

We elected to use a general anaesthetic technique because a regional block was contraindicated by previous spinal fusion. We believed that spontaneous respiration via a facemask was unsafe in this patient who had a history of airway obstruction during sleep. We feared total loss of airway control under anaesthesia, and blind nasal intubation was unlikely to succeed because his neck was fixed in extreme flexion and rotation.

We had planned to use the nasal route for intubation but were unable to pass a 4.5-mm round-tipped red rubber suction catheter through either nostril into the pharynx. Fortunately he could open his mouth sufficiently to allow introduction of the Berman airway. Intubation was further complicated by an enlarged, thickened epiglottis. It is not known whether this is a feature of Proteus syndrome. There was no evidence of cystic lung disease, which would have influenced our approach, and prevented the use both of nitrous oxide and positive pressure ventilation.¹¹ This is

a disease mainly of skeletal tissue rather than muscle, so no risk of malignant hyperthermia was anticipated, and no temperature fluctuations were observed.

Our patient will become progressively more deformed. His episodes of airway obstruction are worrying; indeed, this was the likely cause of death of the Elephant Man.¹² Proteus syndrome will probably be increasingly recognised now that it has been well documented. Those afflicted will present frequently for corrective surgery.

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Forum

Acupuncture and transcutaneous stimulation analgesia in comparison with moderate-dose fentanyl anaesthesia in major surgery

Clinical efficacy and influence on recovery and morbidity

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Summary

The efficacy of acupuncture and transcutaneous stimulation analgesia, supplemented by small doses of fentanyl (mean 1.2 µg/kg, SD 1.7) was compared with moderate-dose fentanyl anaesthesia (mean 22.9 µg/kg, SD 2.8) in 29 patients who underwent surgery for retroperitoneal lymph node dissection. The present study describes the anaesthetic techniques and comparison of haemodynamics, demand for analgesics after surgery, recovery and blood gases, restoration of urinary and bowel functions, convalescence in terms of self-reliance and the postoperative course in respect of fatigue and morbidity. A more rapid return of consciousness, an absence of hypercapnia and a smaller decrease in pH were observed in patients who received acupuncture and transcutaneous stimulation ($p < 0.05$). No clinically relevant disadvantages attributable to the method were demonstrated.

Key words

Analgesia; postoperative.

Acupuncture; morbidity.

Transcutaneous nerve stimulation

The use of acupuncture as an anaesthetic in China has led to its investigation and application in the West. Incomplete analgesia is the main disadvantage of the technique, and limits its use.¹ It has therefore been modified and combined with chemical anaesthetics: conventional drugs are administered to induce sleep, unconsciousness and muscle relaxation, and per- and (or) transcutaneous stimulation is used for pain relief. This technique has been used by anaesthetists in a wide range of situations^{2–4} and its ability to obviate or reduce the need for opioids has been demonstrated intra-operatively and in the early postoperative period.^{5–7} However, a more intensive investigation to evaluate the clinical efficacy of the technique and its influence on recovery and morbidity after a standardised surgical procedure has not been performed previously. The present study was designed to discover whether the reduction in dose of opioids associated with the use of the technique would result in stable peri-operative haemodynamics or an increased demand for postoperative analgesics. In addition, an evaluation was made of peri-operative blood loss and postoperative convalescence and morbidity.

Methods

The study was approved by the hospital Ethics Committee. A total of 29 patients (ASA 1 or 2) scheduled for retroperitoneal lymph node dissection due to a non-seminomatous testicular tumour, were studied. Informed consent was obtained at the pre-operative visit. Those who required

prolonged postoperative artificial ventilation were excluded. Patients and the investigator who acquired postoperative data were not aware of the anaesthetic technique used. Patients were premedicated with diazepam 10 mg orally 90 minutes before induction of anaesthesia.

An intravenous infusion and a radial artery catheter were inserted after arrival in the anaesthetic room. Four electrocardiogram (ECG) electrodes (3M) were placed paravertebrally⁸ and symmetrically between T₄ and L₂ (Table 1) corresponding to the dermatomes of the surgical site (i.e. xiphoid to upper part of pubic bone) and connected to a Doltron ESA 400 stimulator. ECG and arterial pressure were monitored continuously.

Anaesthesia was induced with thiopentone (5 mg/kg) and vecuronium (0.1 mg/kg) was administered. Manual mask ventilation was given (N₂O : O₂ = 6 : 3). Patients were then allocated at random to one of two groups referred to as combined acupuncture and transcutaneous stimulation analgesia (CATA) and moderate-dose fentanyl anaesthesia (MFA). The Doltron ESA 400 was switched on (10 Hz, 40 mA, 0.5% pulse width and 40 volts) if the patient was in the CATA group. Three symmetrical ear points¹ were selected for puncture (Table 1), and the needles were connected to a Chinese-made stimulator WQ-6F and stimuli applied (10 Hz up to 20 mA and a 0.7 msec pulse width). Tracheal intubation was delayed until paravertebral and ear points' stimulation had been applied for 15 minutes, or until 15 minutes after induction. A catheter was then inserted into the right internal jugular vein. A bladder

Table 1. Localisation of body (1–4) and ear (5–7) points used in surgery for retroperitoneal lymph node dissection.

Point	Anatomical location
1. Jueyinshu	3 cm lateral to the lower border of the spinous process of T ₄
2. Geshu	3 cm lateral to the lower border of the spinous process of T ₇
3. Danshu	3 cm lateral to the lower border of the spinous process of T ₁₀
4. Shensu	3 cm lateral to the lower border of the spinous process of L ₂
5. Fe	cavum concha: a U-shaped area superior, inferior and posterior to the point Heart
6. Shenmen	triangular fossa: at bifurcation of superior antihelix crus and inferior antihelix crus
7. Jiaogan	inferior antihelix crus: at junction of inferior antihelix crus and medial border of helix

catheter was inserted before surgery, which started 30 minutes after the start of induction or stimulation. Stimulation was continued until the end of the procedure.

Ventilation was controlled (N₂O : O₂ = 2 : 1) to maintain end-expiratory P_{CO₂} at 5.1–5.3 kPa. Muscle relaxation was provided by a continuous infusion of vecuronium until the peritoneum was closed; the rate (2–6 mg/kg) was determined by 1 or 2 weak contraction responses of the *adductor pollicis* muscle as monitored continuously by supramaximal stimulation of the ulnar nerve through surface electrodes near the left wrist (Myotest). No inhalational agent was used.

The Doltron ESA 400 was not switched on, nor the ear points punctured, if the patient was in the MFA group. Patients in this group received fentanyl (10 µg/kg) as an intravenous bolus 5 minutes before incision and as a continuous infusion at a rate of 5 µg/kg/hour for the first hour and 4 µg/kg/hour thereafter until the surgeon began to suture the skin. Fentanyl 50 µg was given to patients in either group if analgesia at any time was considered to be insufficient, as indicated by either an increase of arterial pressure and (or) heart rate by more than 25% of baseline values, or the presence of autonomic signs such as tears, sweating or salivation. Peri-operative colloids and crystalloids were infused (10–15 ml/kg/hour) to maintain the central venous pressure within normal limits. Blood and fluid losses were assessed, and if necessary, packed red cells were given. Postoperatively, patients received a glucose/saline (3.3%/0.25%) infusion (100 ml/hour) until oral fluid was allowed. No parenteral nutrition was given.

Residual curarisation was determined by nerve stimulation and, if indicated, pyridostigmine (5–10 mg) and atropine (0.25–0.50 mg) were given. Apnoeic patients in whom end expiratory P_{CO₂} was above 10.7 kPa received increments of intravenous naloxone 0.04 mg. Patients were kept in the recovery room for careful observation for at least 90 minutes after extubation. Arterial pressure and heart rate were recorded on arrival, before and after induction, after 15 minutes of stimulation, 1 minute after tracheal intubation, at incision and then at 10-minute intervals in the first hour and at 15-minute intervals thereafter. In the recovery room arterial pressure, heart rate and respiratory rate (RR) were recorded at 30-minute intervals.

Degree of consciousness (can be awakened, disorientated or fully awake) was assessed on arrival in the recovery room, after 1 hour and just before returning to the ward. Arterial blood gases were analysed in duplicate 1, 3 and 5 hours after surgery, while the patient breathed air. The duration of recovery room stay was recorded. The haemo-

globin concentration (Hb), haematocrit (Hct) and assessment of pain using a mixed verbal/visual analogue pain scale of 0–3 (0, no pain, 3 severe pain) were assessed pre-operatively, 1 hour after surgery and again daily at 1300 hours until day 6. The incidences of nausea and vomiting were recorded. Intramuscular nicomorphine (0.1 mg/kg) and prochlorperazine as a 25-mg suppository were available for analgesia and antiemesis respectively. The patient's opinion of the anaesthetic was evaluated as bad, sufficient, satisfactory or good on days 1, 2 and 3. Patients were questioned on each of those days about awareness during surgery or pain in the peri-operative period. Awareness was defined as remembering conversations which occurred during anaesthesia and (or) having experienced unpleasant dreams.

The time between the end of surgery and the removal of the bladder catheter (following the patient's ability to micturate) was assessed. The return of colonic motility (assessed by bowel sounds), the first passage of flatus and the time when fluid and (or) food was first consumed orally were recorded. In addition, daily activities such as getting out of bed, ability to go to the toilet, wash and go for a walk, were noted and patients were asked to assess their fatigue level on a linear visual analogue scale with extremities labelled 1, fit and 10 exhausted. The incidence of any complication and the length of hospitalisation were recorded. A standard 12-lead ECG was recorded daily at 1300 hours until day 6 by the same personnel. ECG abnormalities analysed were: prolonged PR interval (> 0.20 seconds), horizontal ST-segment depression (≥ 1 mm from the isoelectric line), flattened T wave (≤ 1/8 of previous R wave), inverted T wave (isolated inverted T wave on leads III or V₁ were not included) and the presence of a U wave.

Variables measured on admission or before induction served as control. Data are presented as mean (SD) unless stated otherwise. General linear models for repeated measures and Fisher's exact test were applied; *p* < 0.05 was accepted as the level of statistical significance.

Results

Twenty-nine patients entered the study. One patient in the CATA group and three in the MFA group were withdrawn because of excessive peri-operative blood loss of > 5 litres. This led to a violation of the protocol because post-operative artificial ventilation was needed. The data of one CATA patient who did not respond to the stimulation and required a dose of fentanyl which equalled 76% of the mean dose required by MFA patients, although completely assessed, were excluded from analysis. Table 2 shows details of the remaining 24 patients. The two groups of 12 patients were comparable in respect of sex, age, weight, height and durations of anaesthesia, surgery and recovery

Table 2. Mean (SD) values of demographic data, duration of anaesthesia and surgery, and the stay in the recovery room (recovery time).

	CATA	MFA
Sex ratio; M/F	12/0	12/0
Age; years	24.8 (6.3)	30.0 (9.6)
Weight; kg	70.2 (5.6)	78.0 (15.5)
Height; cm	181.7 (3.6)	180.3 (8.2)
Anaesthetic time; hours	4.1 (0.6)	3.8 (0.3)
Surgical time; hours	3.4 (0.6)	3.1 (0.4)
Recovery time; hours	2.2 (0.3)	2.4 (1.3)

There was no significant difference between the groups (ANOVA).

Table 3. Mean (SD) values of peri-operative anaesthetic drug administration and postoperative demand for nicomorphine.

Drug	CATA	MFA
Thiopentone; mg/kg	5.0 (0.3)	5.4 (1.6)
Vecuronium; mg/kg/hour	0.1 (0.04)	0.1 (0.02)
Vecuronium; mg/kg	0.4 (0.1)	0.3 (0.1)
Fentanyl; μ g/kg/hour	0.3* (0.4)	6.3 (1.3)
Fentanyl; μ g/kg	1.2* (1.7)	22.9 (2.8)
Nicomorphine; mg/kg	0.9 (0.5)	0.8 (0.4)

* $p < 0.05$.

room stay. Table 3 shows the peri-operative drug administration and the postoperative requirement of nicomorphine. No differences were found between the groups except for the dose of fentanyl. Four of the 12 patients in the CATA group tolerated surgery without fentanyl, whereas seven of the 12 patients in the MFA group did not need any supplement of fentanyl above the doses required by the protocol. The fentanyl requirement in the CATA group was 4.7% (SEM 1.8; range 0–19.9%) of the mean dose in the MFA group. Naloxone 0.08 mg was given to one MFA patient.

Comparable arterial pressures and heart rates were found in the two groups before surgery (after 15 minutes of stimulation or 15 minutes after induction), after tracheal

intubation and intra- and postoperatively in both groups (Fig. 1). There were significant increases of arterial pressure and heart rate by 21% and 35% (CATA) respectively 19.4% and 32% (MFA) after tracheal intubation. Both arterial pressure and heart rate at incision were lower than baseline ($p < 0.05$) in the MFA group, whereas there was no such decrease in the CATA group; the difference between the groups was significant. Both arterial pressure and heart rate in the MFA group increased shortly after incision to values similar to or greater than those observed in the CATA group. Tachycardia and arrhythmias were not observed in either group.

Arterial pressure in the CATA group was consistently but not significantly lower than that observed in the MFA group ($p = 0.22$) at awakening and during the recovery room stay; heart rate was similar in both groups. The CATA group had a consistently but insignificantly higher RR compared with the MFA group (Fig. 1). All CATA patients were fully awake (Table 4) on arrival in the recovery room as opposed to seven in the MFA group ($p < 0.05$). No patient in the CATA group was disorientated, compared to three in the MFA group. Blood gas results are shown in Figure 2. Both groups had a lower P_{aO_2} than the baseline value throughout the assessment period ($p < 0.01$), but there were no differences between groups. Patients in the CATA group had a normal mean P_{aCO_2} (range: 3.2–5.8 kPa), whereas in the MFA group P_{aCO_2} (range: 4.2–9.0 kPa) was without exception higher than baseline ($p < 0.01$); between the groups, differences existed at 1 and

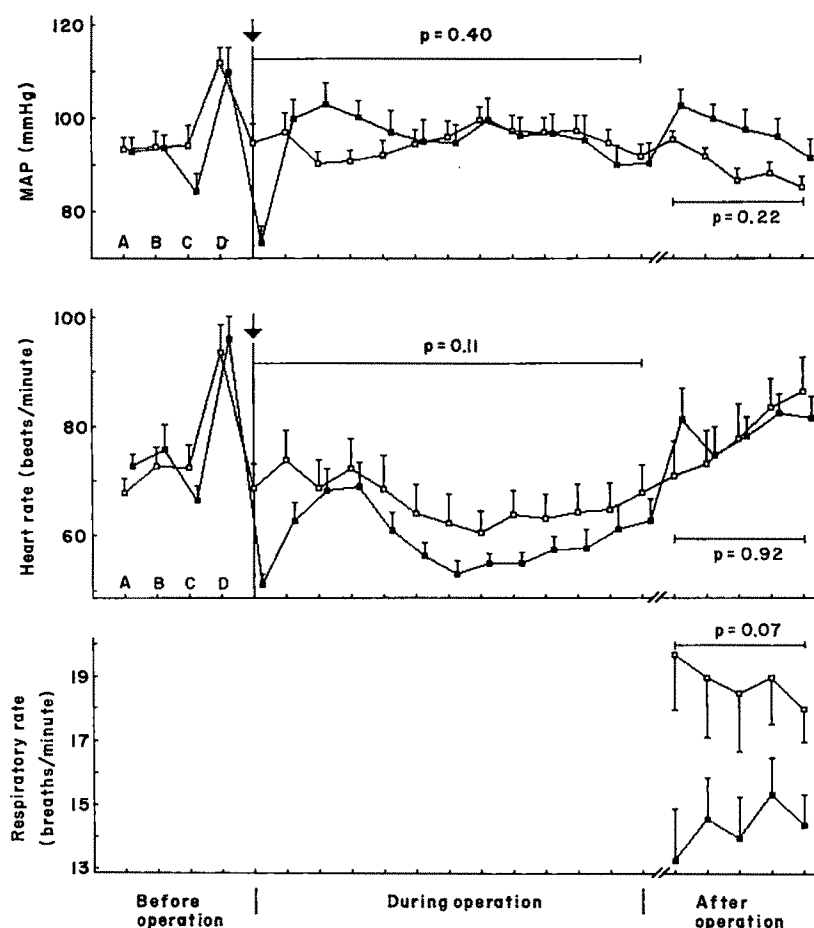


Fig. 1. Mean arterial pressure, heart rate and respiratory rate (error bars equal SEM): $\square$ — $\square$, CATA; $\blacksquare$ — $\blacksquare$, MFA. Arrow indicates the incision time. A = admission, B = 5 minutes after insertion of arterial line, C = after 15 minutes of stimulation, D = 1 minute after tracheal intubation. The significance of the differences from baseline of mean responses during and after surgery are given by p values.

Table 4. Degree of consciousness (number of patients) on arrival at the recovery room (T1), 1 hour later (T2) and at the end of the recovery period (T3).

Consciousness	T1		T2		T3	
	CATA	MFA	CATA	MFA	CATA	MFA
Can be awakened	12	9	12	11	12	12
Disorientated	0	3	0	0	0	0
Fully awake	12	7*	12	11	12	12

* $p < 0.05$ (Fisher's exact test).

3 hours ($p < 0.05$). Both groups had a lower pH ($p < 0.01$) than baseline (CATA range: 7.29–7.45; MFA range: 7.19–7.36); differences between the groups existed at 1 and

3 hours ($p < 0.05$). Values of standard bicarbonate and base deficit were comparable. The fluid balance during operation and in the early postoperative period was comparable between groups. One CATA patient received 2 units, and one MFA patient one unit, of packed red cells during surgery. The values of Hb and Hct one hour after surgery were comparable and significantly lower than baseline: 8.1 mmol/litre (0.7) and 0.39 (0.03) respectively in the CATA group and 8.6 (0.9) mmol/litre and 0.41 (0.05) in the MFA group. The incidences of nausea and vomiting in the recovery room and the first 3 days after operation were comparable in the two groups. Prochlorperazine was given to one CATA patient and five MFA patients on day 1, but this difference was not statistically significant. The total dose of prochlorperazine required was 10.4 mg (16.7) in the CATA group and 19.2 mg (26.1) in the MFA group. No patient complained of recall of pain during operation, but

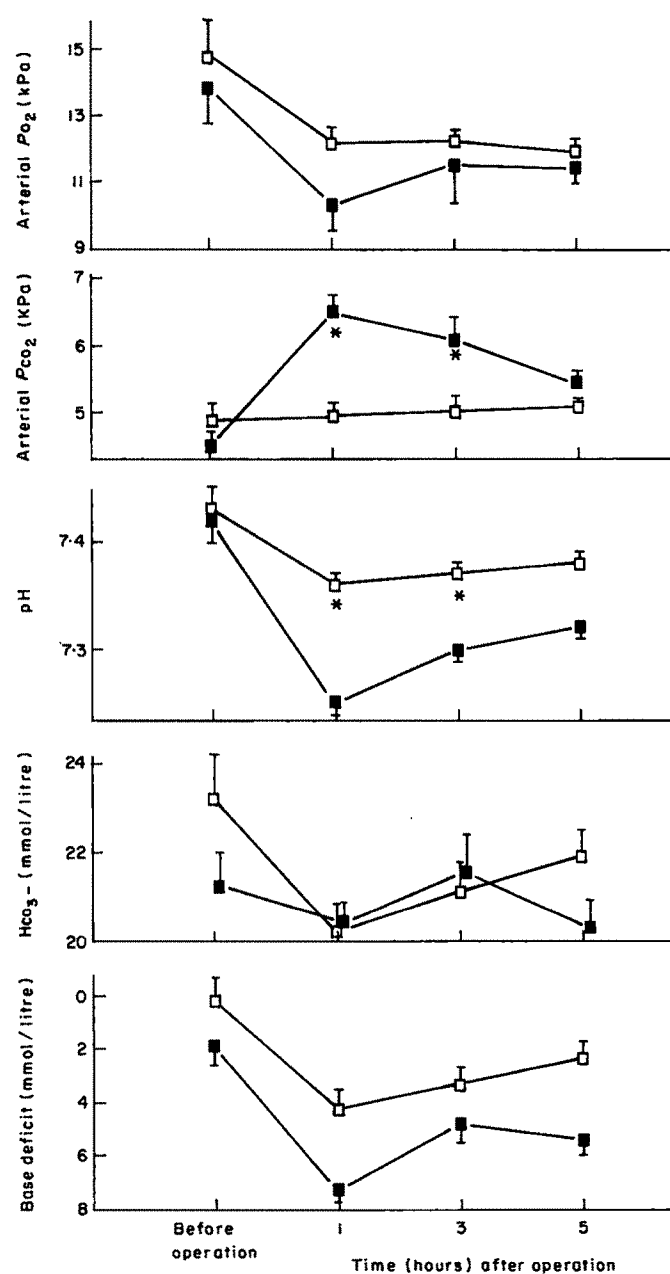


Fig. 2. Arterial blood gases in the early recovery period. For groups and symbols see Figure 1. * indicates statistically significant difference between the groups at various sampling points.

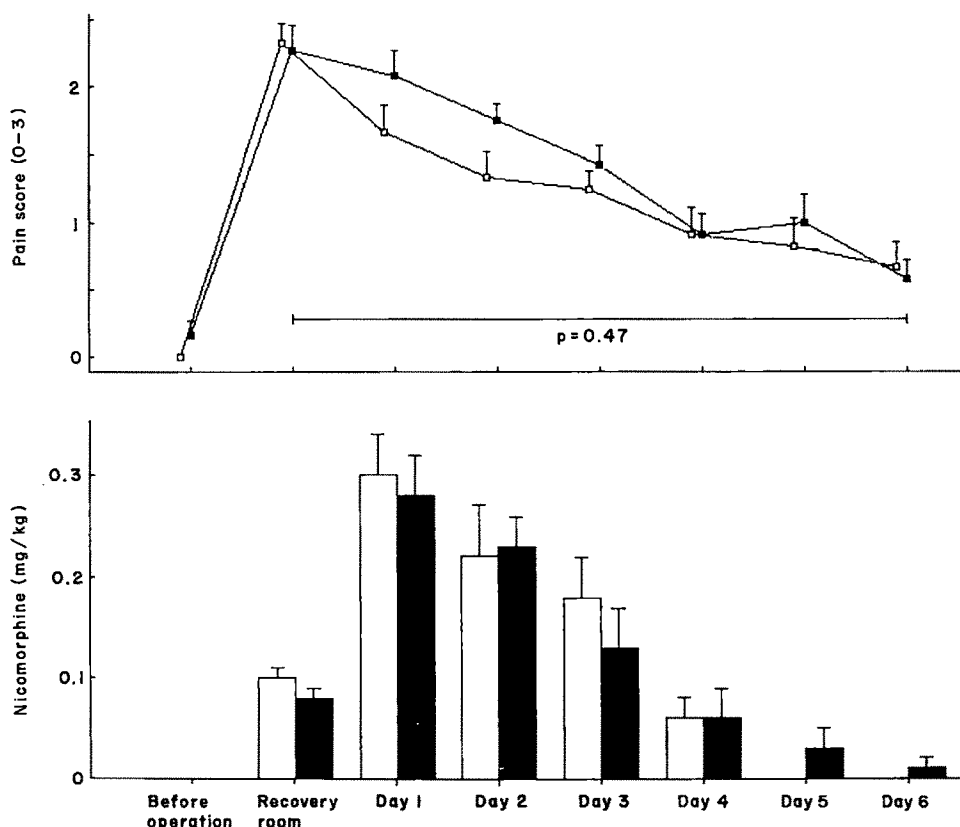


Fig. 3. Postoperative pain score and the demand for nicomorphine. For groups and symbols see Figure 1.

one CATA patient and two MFA patients had awareness of other intra-operative events. These patients judged the anaesthetic technique as sufficient; one of the two patients in the MFA group described the technique as bad and the other as satisfactory on day 3. However, the majority of opinions among other patients were satisfactory and good for both anaesthetic techniques. The postoperative pain scores and the daily requirements of nicomorphine (Fig. 3) did not differ significantly ($p = 0.47$) between the groups. The highest score was recorded one hour after surgery and the highest requirement of nicomorphine was on day 1. None of patients in the CATA group consumed nicomorphine on days 5 and 6.

The bladder catheter was removed after 17.8 (12.9) hours in the CATA group and after 26.3 (10.7) hours in the MFA group; the difference was not statistically significant. Comparable data were found in the two groups concerning the resumption of colonic peristalsis (CATA 39.4 (9.5) hours; MFA 42.1 (19.1) hours), the delay in passing flatus (CATA 51.2 (3.1) hours; MFA 51.3 (14.0) hours), time resumption of oral fluid (CATA 2.8 (0.7) days; MFA 2.6

(0.6) days) and time to restarting food intake (CATA 4.4 (1.5) days; MFA 4.4 (1.9) days). The fatigue score (Fig. 4) and the length of hospitalisation (CATA 9.3 (2.2) days; MFA 10.0 (2.5) days) did not differ between the groups. The fatigue score increased after surgery ($p < 0.05$) and had normalised by day 10 ($p = 0.26$) in both groups. The convalescence in terms of self-reliance was comparable in the two groups.

Temporary ECG signs of myocardial ischaemia (T-wave flattening) occurred in both groups; the highest incidence was recorded on days 2 and 3 (11 and 8 CATA patients; 8 and 8 MFA patients). ST depression was found in one MFA patient on days 1 and 2. No other ECG abnormalities were recorded.

Discussion

It was impossible to carry out the present study in a double-blind fashion, even for a short period until surgical incision. First, the stimulation of ear points is followed quickly by erythematous changes of the ears which can be

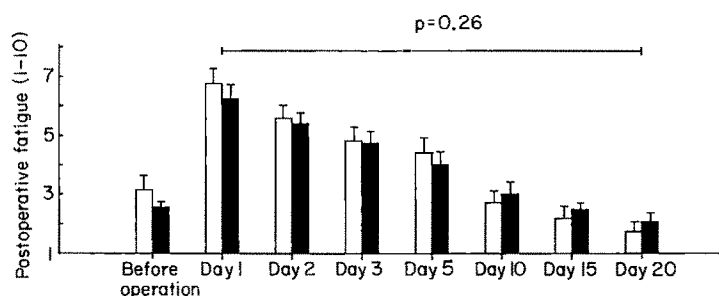


Fig. 4. Postoperative fatigue scores. For groups and symbols see Figure 1.

observed readily. Secondly, stimulation causes a small artefact on the ECG monitor which may be recognised easily. Bias was minimised by ensuring that the study was randomised, that it was performed as single-blind and that the evaluators outside the operating theatre were blinded.

Intubation of the trachea was delayed for 15 minutes to allow time for body- and ear-points' stimulation to take effect. Nevertheless, CATA did not prevent the cardiovascular response. The same phenomenon was found in the MFA group and it was clear that neither 70% nitrous oxide nor its combination with acupuncture and transcutaneous stimulation were sufficient to prevent the pressor response to intubation. Many methods to modulate this response have failed to gain widespread acceptance as a result of either a lack of efficacy, inapplicability for routine anaesthetic practice or complexity and problems inherent in the technique.⁹⁻¹² Frequent traction on the peritoneum, and exploration and mobilisation of abdominal organs and lymph nodes near the vessels and sympathetic ganglia, were reflected by the patterns of MAP and HR in the MFA group; even the bolus of fentanyl given 5 minutes before incision, which caused decreases of MAP and HR (due presumably to vagal effects of fentanyl), did not prevent the continuous increase of MAP and HR in the first 30 minutes after incision. However, patients in the CATA group had a relatively stable MAP and HR over the same period. This stability may have been achieved by a combination of at least two factors: analgesia produced by acupuncture and transcutaneous stimulation and a vagal effect due to stimulation of ear areas innervated by the vagal nerve. However, the overall patterns of haemodynamics in both groups were stable. Earlier data from a controlled study comparing four narcotic drugs in balanced anaesthesia revealed that a potent inhalational agent was required in 23% of patients to maintain stable haemodynamics.¹³ The CATA group required a considerably smaller amount of fentanyl (average total dose 1.2 (1.7) $\mu\text{g/kg}$) than the MFA group (22.9 (2.8) $\mu\text{g/kg}$). It could be suggested that excessive fentanyl was given, particularly since naloxone had to be administered in one patient to restore ventilation. However, we do not believe that the fentanyl dose was excessive because of the changes in haemodynamics during the first 30 minutes after incision and because the maintenance dose of fentanyl given as a continuous infusion was derived from the average requirements for fentanyl for this type of surgery performed under balanced anaesthesia without the use of neuroleptic, benzodiazepine or inhalational anaesthetics. This was calculated to be 100 to 150 μg every 10 minutes. In addition, five of the 12 patients required another supplement of fentanyl in excess of the doses given according to the protocol.

The incidence of a nonresponder in the CATA group is in accordance with previous data.⁴ The requirement for muscle relaxant in the CATA group did not increase, a finding which accords with an earlier study,⁵ although a contradictory result has also been reported.¹⁵ This could be explained by the retrospective design in the first study and the absence of monitoring in the latter.

The demand for fentanyl in the CATA group was less, and this may account for the rapid awakening and the absence of hypercapnia. The latter led, in part, to the lowering of pH in the MFA group. Although arterial PO_2 decreased we did not observe hypoxaemia in either group in contrast to other studies of upper abdominal surgery.¹⁵⁻¹⁷ Acupuncture has been reported to reduce postoperative nausea.¹⁸ This might contribute to the lower need for prochlorperazine on day 1 in the CATA group, the requirement of postoperative analgesics being comparable in the two groups.

Several factors may have contributed to the high inci-

dence of awareness (CATA : 1/12; MFA : 2/12). CATA patients received opioids only on signs of insufficient analgesia, although all denied remembering feeling peri-operative pain, and the use of nitrous oxide-oxygen anaesthesia and opioids in combination with neuromuscular blockade is known to be associated with a high incidence of awareness.¹⁹ The occurrence of awareness in the MFA group indicates that unconsciousness cannot be assured even when this commonly employed technique is used. Awareness is reported to occur in 1 to 4% of cases.¹⁹ A much higher incidence, e.g. 14 from 20 cases, has recently been reported with the use of a routinely applied anaesthetic technique.²⁰ This major variation of reported incidences of awareness is related to the definitions used.

Following laparotomy normal colonic motility is resumed after at least 48 hours. Increased sympathetic activity and the use of opioids are the major causes of prolonged postoperative alteration of colonic motility.²¹ The CATA group may have failed to show a more rapid recovery of bowel function because of the normal postoperative consumption of opioids, although the peri-operative need was low. The small number of patients may account for the failure of the difference in recovery of urinary function to reach a level of statistical significance ($p = 0.06$). The recovery of fatigue score in both groups, while similar, was obviously shorter than that reported in another study.²² Reversible ECG signs of myocardial ischaemia developed postoperatively; similar findings have been reported after cholecystectomy.²³ The recovery might possibly be more rapid in the CATA group, although the significance of this phenomenon is doubtful considering the time course and the great number of comparisons made. It is not clear whether the reported improvement of microcirculation and coronary blood flow due to a reduction of blood viscosity by acupuncture²⁴ contributed to the observed ECG recovery.

In conclusion, the use of acupuncture and transcutaneous stimulation in upper abdominal surgery, although not preventing the pressor response to tracheal intubation, maintained stable haemodynamics with a rapid recovery without hypercapnia. Any other clinically relevant disadvantages attributable to the method, though carefully searched for, were not demonstrated.

Acknowledgment

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Oxygen delivery systems—a comparison of two devices

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Summary

Two low-volume, variable performance oxygen delivery systems were compared in conscious spontaneously breathing volunteers. Oropharyngeal oxygen concentrations were measured during periods of nose and mouth breathing. The systems were studied at oxygen flow rates of 2 or 4 litres/minute. The performance of both systems was similar under the test conditions but the nasal catheter is preferable in terms of cost.

Key words

Oxygen; delivery system.

Hypoxaemia is a frequent occurrence in the immediate postoperative period. It may be subdivided into early hypoxaemia lasting up to 2 hours and late hypoxaemia which may last for several days after surgery. The relationship between hypoxaemia during and after anaesthesia and

postoperative pulmonary function has been discussed at length.^{1,2}

It is common practice to try to prevent hypoxaemia by increasing the concentration of inspired oxygen. Many methods are available and the subject has been reviewed

Table 1. Oropharyngeal oxygen concentrations for each subject.

Subject number	Nose breathing				Mouth breathing			
	2 litres/minute		4 litres/minute		2 litres/minute		4 litres/minute	
	Mask	Catheter	Mask	Catheter	Mask	Catheter	Mask	Catheter
1	28	28	37	36	30	29	36	37
2	26	26	38	37	26	29	40	44
3	29	34	39	35	28	25	39	37
4	31	27	40	41	29	35	42	43
5	31	33	41	38	32	30	40	51
6	29	29	43	45	30	42	45	51
7	27	29	37	38	31	32	41	41
8	28	30	39	36	31	32	42	43
9	26	29	35	38	27	38	33	53
10	30	30	39	36	29	30	33	41

extensively.^{3,4} Oxygen can be delivered to patients by a variety of devices in the recovery area. These can be considered as being fixed or variable performance systems. Several so-called fixed performance devices have been shown not to deliver fixed concentrations of oxygen under all circumstances.⁵ Many anaesthetists administer oxygen postoperatively through variable performance devices allowing for the recognised effect of the breathing pattern on the inspired oxygen concentration.⁶ Facemasks and nasal catheters are examples of variable performance delivery systems. Facemasks seem to be more popular in the UK because it is believed that they are more efficient and more comfortable for the patient; the latter belief possibly may be associated with the results of several studies involving the placement of the nasal catheter in the naso-oropharynx.⁷ Both facemasks and nasal catheters have been used to prevent early postoperative hypoxaemia with equal success.⁸ The use of nasal catheters with a foam collar at the distal end allows the catheter to be located comfortably in the anterior nares and makes the system more acceptable to the patient.

The recovery room in most hospitals deals with large numbers of patients each year and oxygen delivery systems must be not only effective but also inexpensive. The present study was implemented to compare the oxygen concentration in the oropharynx during breathing of low-flow oxygen via a nasal catheter or a low-volume facemask.

Methods

Ten conscious volunteers were studied. All were graded as ASA 1 or 2 and none had an upper respiratory tract infection or nasal airway obstruction. The concentration of inspired oxygen in each subject was measured in the oropharynx during oxygen administration via a low-flow variable performance facemask (Intersurgical*) followed by oxygen administration via a nasal catheter situated in the anterior nares and held in place by a foam collar (Unoplast oxygen catheter with compress†).

A 10-gauge cannula was inserted through the nose after administration of topical anaesthesia, until the tip was situated approximately 10 mm below the uvula when viewed through the mouth. The nasal oxygen catheter was located in the opposite nostril when in use. A fine catheter was positioned 5 mm inside the nostril on the same side as the sampling catheter for the purpose of monitoring the

end-tidal CO₂ concentration. This catheter was located just inside the mouth during mouth breathing.

The sampling catheter was connected to a Servomex oxygen analyser (model 570A). Calibration was performed before use using oxygen-free ('White spot') nitrogen and air. The end-tidal CO₂ was recorded using a Gould capnograph mark 3, calibrated with air and certified 5% CO₂ in air, and the output printed on a Hewlett Packard pen recorder (model 7402A). Oxygen from a calibrated flowmeter was delivered to each subject at 2 or 4 litres/minute through either the facemask or nasal catheter. Recordings were made during nose breathing and mouth breathing. Four readings were taken at one-minute intervals 3 minutes after a steady inspired oxygen concentration and end-tidal carbon dioxide concentration had been achieved and the mean was taken as the inspired oxygen concentration. Results were compared for the two delivery systems using paired *t*-tests with no allowance made for multiple comparisons.

The subjects were asked at the end of the experiment if they found one system more comfortable than the other.

Results

The measured oropharyngeal oxygen concentrations for the 10 volunteers are shown in Table 1.

The mask and nasal catheter produced a mean oropharyngeal oxygen concentration of 28.5% and 29.3% respectively during nose breathing at an oxygen flow of 2 litres/minute; at 4 litres/minute the corresponding mean oropharyngeal oxygen concentrations were 38.8% and 38.0%. There were no significant differences between the mask and catheter at either flow of oxygen, although both systems produced significantly higher oropharyngeal oxygen concentrations at 4 litres/minute than at 2 litres/minute ($p < 0.001$).

The mask produced a mean oropharyngeal oxygen concentration of 29.3% and the nasal catheter 32.2% during mouth breathing at an oxygen flow of 2 litres/minute. The mean concentration achieved by the catheter was higher, but there was a wide range of results and the difference was neither statistically nor clinically significant. The mask produced a mean oropharyngeal oxygen concentration of 39.1% and the nasal catheter 44.1% during mouth breathing at an oxygen flow of 4 litres/minute. Again the mean concentration achieved by the catheter was higher, and the range wider; there was a statistical difference ($p < 0.05$) which probably has no clinical significance. Both systems produced significantly higher oropharyngeal oxygen concentrations at the higher oxygen flows ($p < 0.001$).

The mean, range and 95% confidence intervals of the

*Crane House, Gould Road, Twickenham, Middlesex.

†Wykeham industrial estate, Moorside road, Winchester, Hampshire.

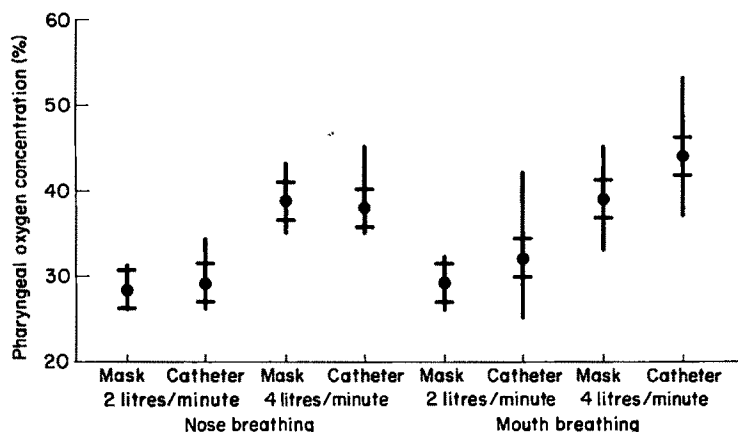


Fig. 1. The range of pharyngeal oxygen concentrations measured in each group, together with the means (●), 95% confidence intervals (=) and range (|).

pharyngeal oxygen concentrations are shown in Figure 1 and the results of the paired *t*-tests in Figure 2.

Both systems reliably delivered acceptable oxygen concentrations which are suitable for clinical administration and which are close to the delivered concentrations detailed by the manufacturers. Eight of the subjects found the nasal catheter more comfortable when asked directly, while the other two subjects found both systems equally comfortable.

Discussion

The aim of postoperative oxygen therapy is to minimise hypoxaemia. The use of a variable performance device is satisfactory in the majority of patients provided that an acceptable minimum oxygen concentration is delivered. However, in some patients the delivery of a precise concentration is desirable and the use of a fixed performance device is indicated. The oxygen delivery system may be assessed on the grounds of efficiency, patient acceptability, simplicity and cost effectiveness.

The range of results for each variable tested in our study was wide and was more marked with the nasal catheter than with the facemask. This large interpatient difference has been reported before.⁷ It may be accounted for by differences in the breathing pattern (although this was minimised by maintaining the end-tidal CO_2 within the normal range), and by minor anatomical differences resulting in turbulent flow or channelling of oxygen.

A minimum oropharyngeal oxygen concentration of 33% was obtained using the mask and 35% with the nasal

catheter when an oxygen flow of 4 litres/minute was used. The minimum concentration was 26% with the mask and 25% with the catheter at a flow of 2 litres/minute. These small differences between the devices are not of any clinical significance. However, the delivered concentration does depend upon the inspiratory flow rate,^{5,6,10} although reduction in the inspired concentration of oxygen due to increased inspiratory airflow may be compensated by increasing the flow of oxygen to the delivery system.

The choice of system is important. A nasal catheter is not suitable in patients with nasal obstruction or in those who have undergone nasal surgery. In this study, 80% of the subjects found the nasal catheter was more comfortable than the facemask, and the fixation of the catheter by means of the foam collar is easier than the elastic head band of the mask. Flexion of the head and neck is not required to fix the device and observation of the patient's colour is easier. Nasal delivery systems may become uncomfortable at oxygen flows greater than 4 litres/minute.

The relative cost of two equally efficient systems is significant when the purchase of large numbers is considered. Nasal catheters are the least expensive method of oxygen administration,⁸ and it is interesting to speculate why their use is not more widespread. This may be because in some studies the catheter was threaded beyond the nose and into the nasopharynx, an uncomfortable and potentially traumatic procedure. At the time of writing (January 1990) the cost of the Unoplast 10-gauge nasal oxygen catheter with compress is 26 pence; the cost of the Intersurgical facemask is 44 pence. The use of facemasks in

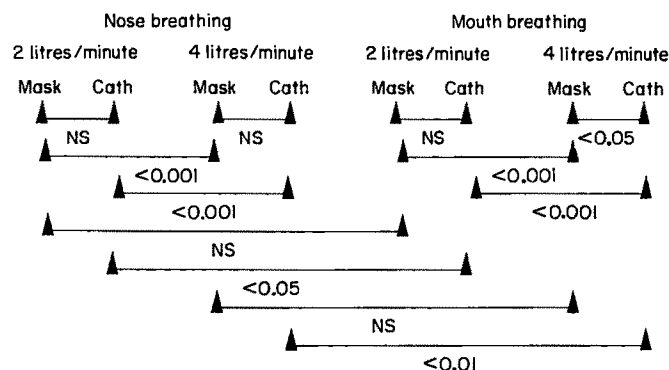


Fig. 2. The probabilities (calculated using paired *t*-tests) that the differences between the means of each group are due to chance. $\alpha = 0.05$, and no correction for multiple *t*-tests has been made.

a recovery area which provides oxygen therapy for 10 000 patients per year is £4400. The cost would be £3320 if nasal oxygen catheters were used on 60% of patients, a saving of £1080 (24.5%). This saving would be greater if the use of nasal catheters was implemented in other departments throughout the hospital.

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Postoperative analgesia after triple nerve block for fractured neck of femur

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Summary

Fifty patients with fractured neck of femur that required surgical correction with either a compression screw or pin and plate device were randomly allocated to receive one of two anaesthetic techniques, general anaesthesia combined with either opioid supplementation or triple nerve block (three in one block) with subcostal nerve block. The nerve blocks significantly reduced the quantity of opioid administered after operation; 48% of these patients required no additional analgesia in the first 24 hours. Plasma prilocaine levels in these patients were well below the toxic threshold, and peak absorption occurred 20 minutes after the injection. No untoward sequelae were associated with the nerve blocks.

Key words

Anaesthetic technique, regional; triple nerve block, subcostal nerve block. Analgesics, local; prilocaine. Surgery; orthopaedic.

Fracture of the neck of the femur is a common injury especially in elderly females and is associated with significant morbidity and mortality. The majority of the pain before surgery originates from the bone, periosteum and local muscle spasm, and is alleviated by internal fixation with either pin and plate devices or the use of compression screws. The pain after operation is from the incision and tissue trauma during surgery. The hip joint, upper femoral shaft and area of skin incision are supplied principally by branches of the lumbar plexus; the femoral nerve, obturator nerve and lateral cutaneous nerve of the thigh. An area of skin immediately above the greater trochanter may be supplied by the subcostal nerve (T_{12}).

These nerves, excluding the subcostal, may be blocked by the injection of a sufficiently large volume of local anaesthetic agent into the sheath that surrounds the femoral nerve just below the inguinal ligament, a method described by Winnie¹ as the inguinal paravascular technique of lumbar plexus anaesthesia, perhaps better known as the 'three in one block' or 'triple nerve block'. Winnie recom-

mended a minimum volume of (patient height in inches/3) ml to ensure a predictable block of all three nerves, although other workers have suggested that a larger volume may be required.

A prospective, randomised study was undertaken to evaluate the use of the triple nerve block combined with subcostal nerve block for postoperative analgesia in patients undergoing surgical correction of their fractured neck of femur. Plasma prilocaine concentrations were measured to assess the absorption of the local anaesthetic agent from this site of injection.

Methods

The study was approved by the local ethics committee. Fifty consecutive patients over the age of 60 years with intertrochanteric fractures of the neck of femur that required surgical correction with either compression screw or pin and plate devices were enrolled into the study after the patients had been resuscitated and written informed

consent obtained. The following patients were not studied: those with an absolute contraindication to a regional technique, those with an allergy to local anaesthetic agents or those who had systemic disease that indicated an alternative method of anaesthesia.

The patients were randomised, by choosing an unmarked envelope, into two groups, namely general anaesthesia or combined general anaesthesia, triple nerve block and subcostal nerve block. Pre-operative analgesia with papaveretum was continued before surgery and no additional premedication was prescribed. No patients received nonsteroidal anti-inflammatory agents.

Baseline records of pulse rate and arterial blood pressure were made in the anaesthetic room and an intravenous infusion established. Group 1 (25 patients) received etomidate 0.2 mg/kg for induction of general anaesthesia. Anaesthesia was maintained with isoflurane in 50% nitrous oxide and oxygen and the patient breathed spontaneously through a facemask via a Lack system. Analgesia was provided with alfentanil in incremental doses of 0.25 mg.

Group 2 (25 patients) received a triple nerve block and subcostal nerve block before the induction and maintenance of general anaesthesia as in group 1. The method used was an adaptation of Winnie's inguinal paravascular technique of lumbar plexus anaesthesia,¹ in that it was performed using an insulated regional block needle (Braun Stimuplex, 22 gauge) and a nerve stimulator. The line of the inguinal ligament and the femoral artery were marked. Under aseptic conditions the needle was inserted 1 cm lateral to the artery and 1 cm below the inguinal ligament in a cephalad direction at 45° to the skin. It was manipulated until a motor response was observed in the motor area of the femoral nerve with a current of less than 1 mA. Thirty-five ml of 0.75% plain prilocaine were injected after negative aspiration while digital pressure was applied distal to the needle to improve cephalad spread.

A further 8 ml 0.75% plain prilocaine were injected along the iliac crest to block the subcostal nerve. Venous blood for plasma prilocaine levels in the initial 12 patients of group 2 was taken from an indwelling cannula at 0, 5, 10, 15, 20, 25, 30, 45 and 60 minutes after the end of the injection of prilocaine. Concentrations were measured using gas chromatography.²

The anaesthetist made 5-minute recordings intra-operatively of arterial blood pressure, pulse rate and any electrocardiographic changes. The concentration of isoflurane and the dosage of alfentanil were adjusted according to the patient's response to surgery, and the criteria used were a 20% change in heart rate or arterial pressure. Any episodes of hypotension, defined as a 30%

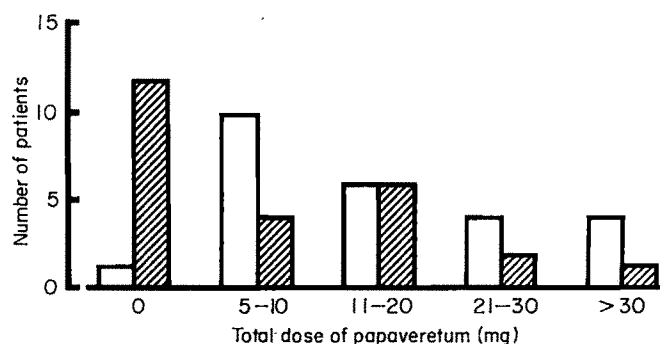


Fig. 1. Total dose of papaveretum in the first 24 hours after surgery. Open columns, group 1; filled columns, group 2 (nerve blocks).

reduction in pre-induction mean arterial pressure that occurred for longer than 5 minutes, were recorded.

The recovery time from discontinuation of the isoflurane after operation to eye opening was recorded. All patients were prescribed intramuscular papaveretum 0.2 mg/kg; administration was at the discretion of the nursing staff who were unaware of the patient's group. All the patients had their skin prepared and an elastoplast placed over the possible injection site to minimise bias.

The operating theatre recovery sister and ward staff, who were blind to the patient's group, were asked to assess the quality of analgesia after operation. The grades were good, no significant pain with movement; equivocal, pain with movement but not at rest; and poor, pain at rest. All patients were interviewed 24 hours after their operation, and a record made of the time and quantity of analgesia administered.

The results were analysed using Student's *t*-test (recovery times), Chi-squared test (incidence of hypotension) and Wilcoxon's rank sum test (percentage of isoflurane). Significance was taken as $p < 0.05$.

Results

Table 1 shows that there was no significant difference between the two groups with respect to age, gender and ASA classification or the type of operation and grade of surgeon.

The insertion of the nerve blocks did not significantly increase the total anaesthetic time. Patients in group 2 required lower maintenance concentrations of isoflurane, but the difference was not significant (group 1 median 1%, range 0.5%–2%; group 2 median 0.5%, range 0–1%).

Table 1. Patient data.

	Group 1 (n = 25)	Group 2 (n = 25)
Gender; males/females	2/23	4/21
Mean age; years (SD)	81 (8.85)	81 (10.1)
range	62–94	63–96
ASA classification		
I	5	5
II	8	8
III	11	10
IV	1	2
Total anaesthetic time; minutes (SD)	83.4 (25)	72.1 (18.2)
Recovery time; minutes (SD)	11.7 (5.5)	7.6 (3.5)*

* $p = < 0.05$.

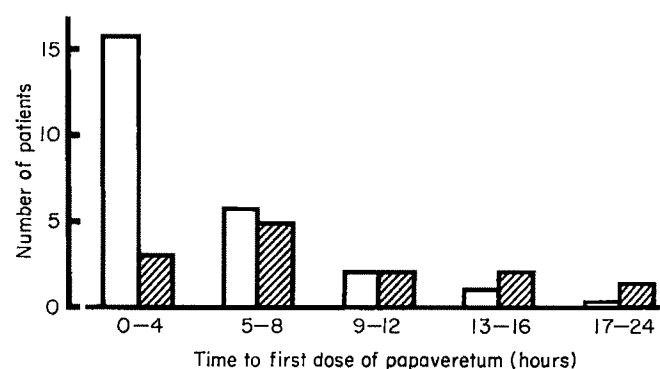


Fig. 2. Time to the first dose of papaveretum. Open columns, group 1; filled columns, group 2 (nerve blocks).

Table 2. Assessment of postoperative analgesia in each group.

	Group 1 (n = 25)	Group 2 (n = 25)	Significance
<i>Recovery Staff</i>			
Good	3	19	< 0.01
Equivocal	6	4	ns
Poor	15	1	< 0.01
<i>Ward Staff</i>			
Good	5	18	< 0.01
Equivocal	6	4	ns
Poor	13	2	< 0.01

Recovery times were significantly shorter in those who received the nerve block ($p < 0.05$). The patients in group 2 received no alfentanil, but all patients in group 1 received alfentanil in a dose range of 0.5–1 mg. There was no difference in episodes of hypotension between the two groups, 14:10 in groups 1 and 2 respectively.

Twelve patients with the nerve blocks required no analgesia in the first 24 hours after operation compared with two patients in group 1 ($p < 0.01$; Fig. 1). Patients in group 2 also had significantly fewer injections in the first 24 hours (44 compared to 19, $p < 0.05$).

There was a significant difference in the number of patients who received analgesia in the first 4 hours from the induction of anaesthesia ($p < 0.01$; Fig. 2).

The quality of analgesia as recorded by both the recovery and ward staff is shown in Table 2. The number of patients described as having good analgesia was significantly greater in group 2 both in the recovery room and at 24 hours.

Figure 3 shows the mean and standard deviation of plasma prilocaine concentrations at each sampling time. The range of the results is wide but the individual peak concentrations occur no later than 25 minutes after injection and have reached a plateau or are declining by 60 minutes. All the plasma prilocaine concentrations (maximum 3 µg/ml), are below the suggested threshold for toxicity for prilocaine of 6 µg/ml.³

There were no untoward sequelae associated with the nerve blocks. One patient (in group 1) died during the first 24 hours after operation and had received no analgesia.

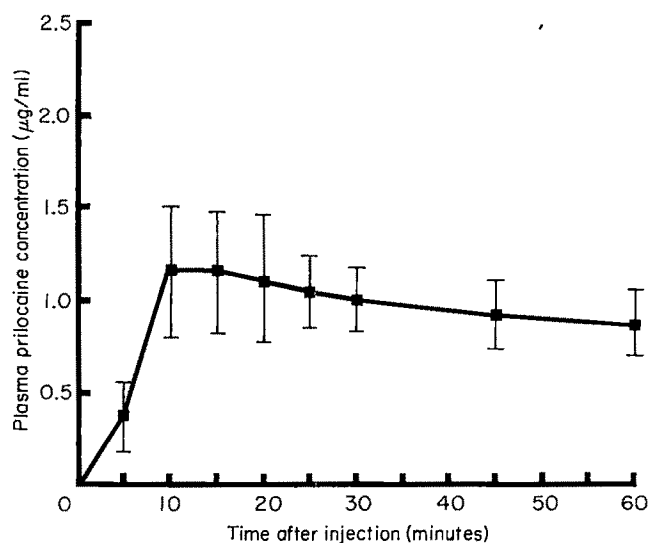


Fig. 3. Mean (SD) plasma prilocaine concentrations.

Discussion

Our study shows that the use of triple and subcostal nerve blocks during the surgical correction of fractured neck of femur significantly reduced the perioperative anaesthetic requirements and resulted in more effective postoperative analgesia; 48% of patients required no analgesia in the first 24 hours. The triple nerve block was simple to perform in comparison with other techniques of regional anaesthesia, the patient could lie supine with skin traction in position, and the procedure was well tolerated in the awake patient.

Surgical correction of intertrochanteric fractures does not involve disarticulation of the femoral head from the acetabulum, which receives part of its innervation from the sciatic nerve. Therefore the triple nerve block with subcostal block would appear to provide adequate analgesia for this type of surgery. The posterior compartment of the thigh is also innervated by the sciatic nerve and may be encroached on by surgical retraction, but despite this theoretical disadvantage no problems were experienced.

The anaesthetist was not blind to the patient's group because the principal aim of the study was to compare postoperative analgesia. Numerous studies have successfully used linear analogue scales, although earlier studies have also shown them to be unsatisfactory in this age group of patients especially after general anaesthesia.⁴ Therefore, in view of the mental state of the patients, an observer assessment was used.

A significant difference was seen after operation despite the apparent imprecise method of assessing the quality of analgesia. This was most evident in the first 4 hours, and represents the relatively short acting effect of prilocaine offset against the short clinical action of alfentanil. Prilocaine was chosen since it was thought to be the safest agent in this elderly population. The plasma levels have demonstrated that this is a safe technique, with peak absorption levels well below the toxic threshold. The rate of decline from peak concentrations was slow and this may be because of continuing absorption from the femoral sheath or the result of decreased clearance in this elderly population. The duration of analgesia may be further increased by the use of bupivacaine.

In summary, the described technique is a safe and convenient method of reducing the perioperative anaesthetic requirements and alleviating the need for opioid analgesics in a significant number of patients during the first 24 hours after surgical correction of fractured neck of femur.

Acknowledgments

We offer thanks to the surgeons, theatre and ward staff of the Royal Hallamshire Hospital for their assistance with this study and to Miss A. Greenaway for her help with the analysis of the plasma prilocaine concentrations.

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Rhabdomyolysis and operating position

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Summary

Rhabdomyolysis during routine surgery was studied in three groups of patients who had surgery, with limited trauma to muscle, in the lateral and supine positions, and prone on the spinal frame. A range of blood tests was performed (before surgery, and on the first, third and seventh day after operation). These showed that a creatine kinase increase in the 24 hours and the early appearance of myoglobin in the serum were the best indicators. Rhabdomyolysis was associated with the lateral position and long lasting surgery. No blood test before surgery was of any predictive value.

Key words:

Muscle skeletal; injury.

Position; effects.

Acute renal failure is occasionally found after surgery. Clinical evidence of surface oedema and necrosis in patients placed prone for prolonged surgery, suggested to us that an investigation should be performed as to whether it is possible to perform any test before operation to predict peri-operative rhabdomyolysis.

Patients and methods

Thirty-six adults were investigated in three groups. The first group ($n = 13$) was in the lateral position, the second was prone on the spinal frame, and the third was supine. The first two groups underwent hemilaminectomy for disc prolapse, the third carotid endarterectomy. Muscle trauma was minimal; the skin incision was 4–8 cm.

The following laboratory tests were performed on the day before surgery, and on the first, third and seventh postoperative days: blood urea, uric acid, creatinine, calcium, potassium, hydrogen ion concentration, magnesium, phosphorus, myoglobin, aldolase, creatine kinase, lactic dehydrogenase, β hydroxybutyrate dehydrogenase, aspartoaminotransferase, creatinurea and daily urine volume.

Anaesthesia was induced in all patients with thiopentone, and maintained with droperidol, fentanyl and nitrous oxide 60%; neuromuscular block was with pancuronium (monitored using a Datex relaxograph). Patients were covered with Thermo-Drapes R to avoid heat loss and ventilation of the lungs was adjusted to produce a P_{aCO_2} of 4.0–5.0 kPa.

Those subjects who shivered at the moment of recovery were not included in the study. After surgery, 30–40 ml/kg/24 hours of dextrose-saline was given to all patients.

Statistical analysis was performed with the Wilcoxon test by separate groups, and for the whole population by a stepwise test.

Results

The anaesthetic and time for surgery were similar for all three groups. Patient characteristics were similar, except for increased age in the third group (Table 1). The earliest indices of rhabdomyolysis were the appearance of myoglobin in the serum and an increase in aldolase and creatine kinase. There was an increase in serum aldolase on the third postoperative day in the lateral position, but this was not statistically significant (Table 2). Serum myoglobin had a peak on the third day after surgery and thereafter decreased. Creatine kinase had a peak on the first day after surgery, and returned to pre-operative values on the third day (Fig. 1). The increases of creatine kinase and myoglobin are more evident in relation to the lateral position, and the duration of the surgery (Table 3).

Discussion

This study investigated patients who underwent elective surgery with minimal muscle damage. There were significant differences in the patients' age (Table 1). Drugs that

Table 1. Patient and anaesthetic data. Values are expressed as mean (SD).

	Group		
	Lateral decubitus ($n = 13$)	Prone ($n = 13$)	Supine decubitus ($n = 10$)
Age; years	47 (12)	42 (9)	61 (7)
BSA; sq m	1.9 (0.1)	1.8 (0.2)	1.8 (0.1)
Surgery time; hours	3.4 (1.4)	2.9 (0.7)	3.5 (0.9)

Table 2. Aldolase course before and after surgery. Values are expressed as mean (SD).

	Group		
	Lateral decubitus (n = 13)	Prone (n = 13)	Supine decubitus (n = 10)
A*(U)	2.80 (1.23)	2.32 (0.46)	2.37 (0.56)
B*(U)	3.18 (1.14)	2.93 (1.04)	2.32 (0.42)
C*(U)	4.11 (2.75)	2.85 (0.75)	2.73 (0.86)
D*(U)	3.02 (2.55)	2.31 (0.39)	2.89 (0.93)

* A, B, C, D = Sample before surgery, 1, 3, 7 days later.

Table 3. Correlation between creatine kinase and myoglobin with other parameters.

Variables	R. multip.	p
Creatine kinase		
lactic dehydrogenase (A)*	0.58	0.001
aspartoaminotransferase (B)*	0.65	0.006
Creatine kinase (B)*		
Surgery time	0.66	0.001
β hydroxybutyrate dehydrogenase (C)*	0.66	0.006
Serum myoglobin (A)†		
Surgery time	0.45	0.005
Serum myoglobin (B)†		
β hydroxybutyrate dehydrogenase (C)*	0.43	0.009

* A, B, C = Sample before surgery and 1 and 3 days later.

† A, B = Serum myoglobin 1 and 3 days after surgery.

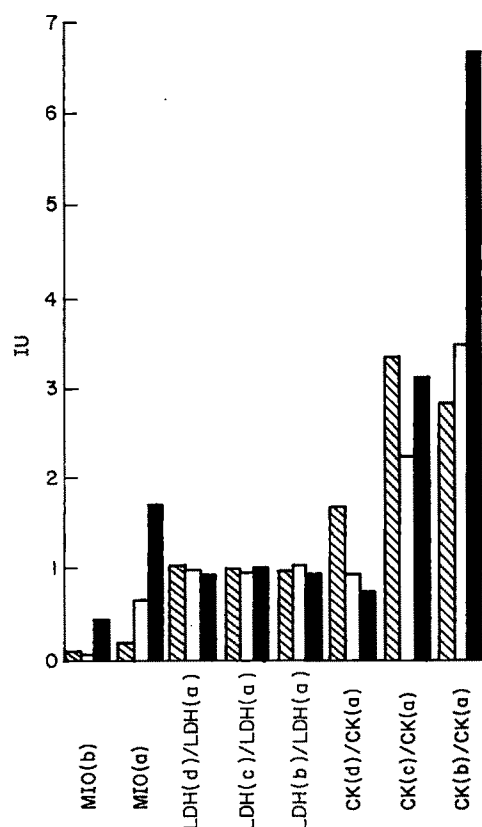


Fig. 1. The increase in creatine kinase (CK), lactic dehydrogenase (LDH) and serum myoglobin (MIO) when compared with the values before operation. ■, lateral decubitus; □, prone; ▨, supine. (a) refers to values before operation; (b), (c), and (d) refer to 1, 3, and 7 days after surgery, respectively.

could cause muscle damage, such as halothane¹ or suxamethonium² were avoided. Droperidol appears only to have the side effect of rhabdomyolysis when used in the long-term treatment of psychosis,³⁻⁵ especially if the patients are malnourished and take other potentially toxic drugs. Other causes are congenital enzyme defect⁶ and direct surgical trauma. Studies have shown necrosis of muscle after prolonged surgery that required the patient to be in anatomically abnormal positions.⁷⁻¹⁰ The pathogenesis of the compartment syndrome involves direct local muscle pressure as the precipitating factor.¹¹ Necrosis of muscle, and loss of capillary wall integrity in the presence of a normal vascular supply will lead to transudation and the development of massive oedema within the compartment. This oedema, in turn, produces pressure within the compartment, which occludes the blood supply and increases the ischaemic necrosis in a vicious circle.⁷ The syndrome can be complicated by both defects in neuromuscular function, and rhabdomyolysis, with or without concomitant renal failure.⁶

This study has implicated lateral decubitus (Fig. 1) as the position most likely to give rise to rhabdomyolysis, while our initial clinical impressions, based on the observations of local oedema and cyanosis would have led us to suspect the prone position. We have, however, observed that the spinal frame, with its padding and minimal body contact, protects against these sequelae. Serum myoglobin and creatine kinase are the earliest indices of rhabdomyolysis (Fig. 1) while the increase in aldolase seen on the third post-operative day is not significant (Table 2). We have also shown that rhabdomyolysis is related to the duration of the surgery (Table 3). We were unable to detect any predictive factor in the blood tests before surgery of subsequent rhabdomyolysis.

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A district-wide anaesthetic audit

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Summary

A district-wide anaesthetic audit has been implemented by the Derby anaesthetic department. Data for every anaesthetic are collected on a specially designed audit form which is read into a computerised database using an optical mark reader. The implementation of the audit, problems encountered and some benefits realised are described.

Key words

Anaesthesia; audit.

Education; training.

The concept of clinical audit is not new and many departments have been auditing their practice for years. We have had a clinical audit of obstetric anaesthesia in Derby for 9 years. Recently, clinical audit has become a mandatory requirement in most specialties, prompted by the initiatives of the Government's White Paper and support from the Colleges.^{1–3} Accordingly our department has undertaken to audit our theatre activity continuously on a District-wide basis.

Methods

The Derby anaesthetic department provides anaesthetic services for the whole of the Southern Derbyshire Health Authority (SDHA), one of the largest nonteaching Health Authorities in England. Approximately 25 000 anaesthetics are administered annually by 46 anaesthetists working at 17 sites in eight hospitals. It was our intention that every anaesthetic given would be included in the audit.

After due consultation, an assurance of cooperation was obtained from all colleagues. Audit forms containing an agreed data set were designed and made available in anaesthetic rooms at every site in the District and one form was completed for each anaesthetic administered. They were designed to be easy to use, and were divided into boxes that required simple ticks to indicate a positive entry. Completed forms were returned daily to the anaesthetic office where a secretary transferred the data into a computerised database.

It was soon realised that the manual method of data entry was very inefficient and to overcome this problem special forms have been designed which are read directly

into the database using an optical mark reader (OMR). Each form took approximately 2 minutes to be entered using the keyboard method of data entry. The OMR has speeded up data capture significantly and although it is capable of reading approximately one form per second it takes a little longer in practice because of simple errors of omission and of accuracy. However, it takes a fraction of the time compared to conventional data input methods.

Our hardware configuration consists of an IBM (80286) compatible microcomputer, two floppy disk drives, a 60 MB hard disk drive and an inkjet printer. The software, written by the author using a commercial package, is designed to be used by a secretary and involves minimal key strokes. It is menu-driven with extensive error-trapping. Data may be entered either manually or automati-

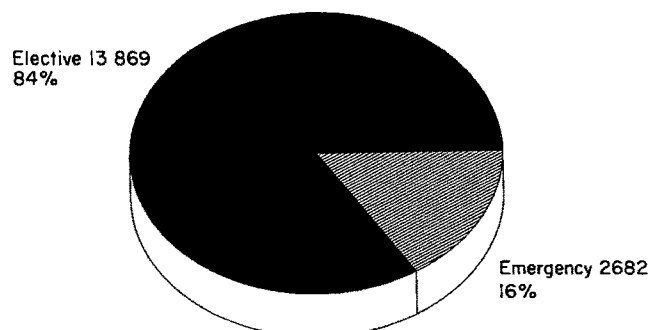


Fig. 1. Number of anaesthetics in units 1 and 2 from June 1989 to February 1990 (total 16 551).

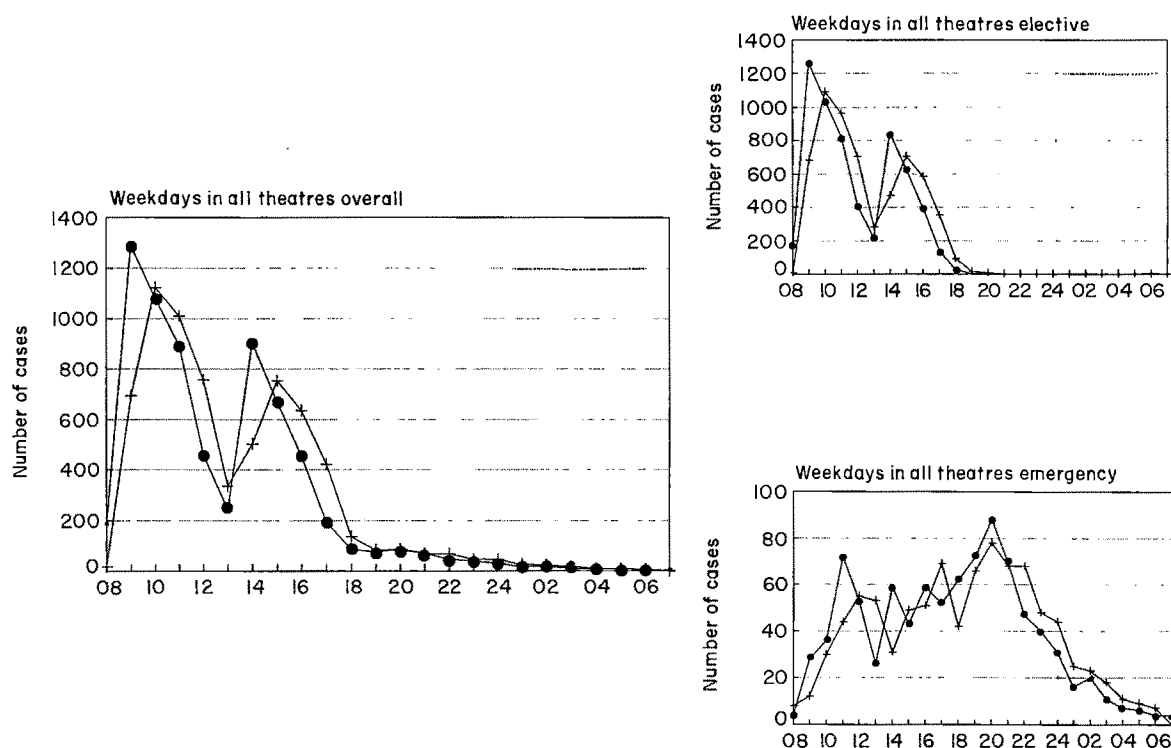


Fig. 2. Work distribution over 24 hours.—●—, start time; +, end time.

cally and sufficient functions are provided to allow database management whilst preserving data integrity. The report generator option allows generation of raw numbers from any datafile and may be used to reflect the activity of the department as a whole, or of an individual over a given period of time, as well as activity at any given location.

Results and discussion

Funding. Implementation of our audit was delayed for approximately 18 months because of lack of funding. We were able to purchase equipment and secretarial time when funding became available. Our ability to produce worth-

while results and willingness to be accountable for the funds allocated has changed our management's attitude towards us and we now find them much more forthcoming.

Compliance. It was essential before embarking upon a project of this size that compliance from all members of the department was obtained. A few anaesthetists had expressed reservations about the concept of audit and about the amount of extra work it would entail, but they rapidly accepted its value. Compliance from Operating Department Assistants (ODA) and theatre nursing staff was equally important and full cooperation was forthcoming only after appropriate feedback.

Office procedures, form filling and data capture. It was

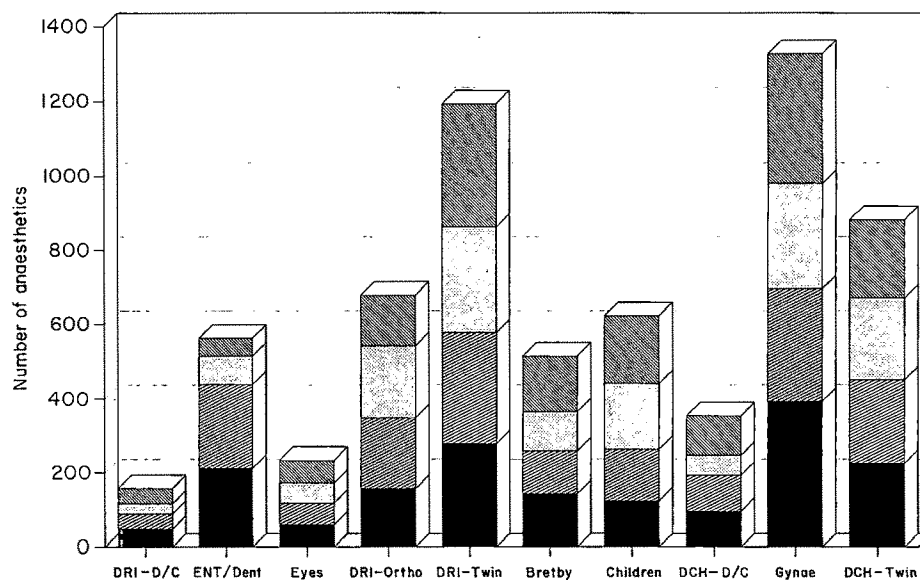


Fig. 3. Theatre workload at various sites (1989). ■, June; ▨, July; ▩, August; ▪, September. DRI, Derby Royal Infirmary; Bretby, Bretby Orthopaedic Hospital; DCH, Derby City Hospital.

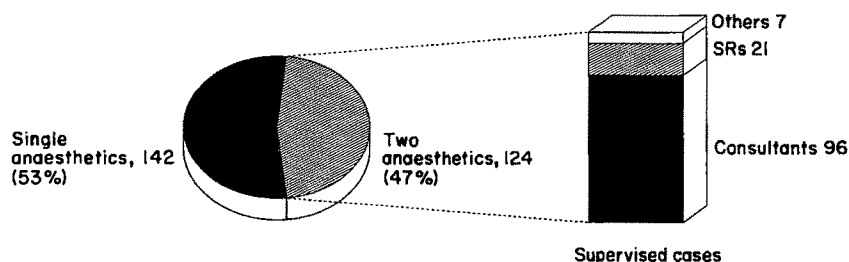


Fig. 4. Supervision of a trainee.

important that the quality of input be as accurate and complete as possible in order to ensure a high quality of output from our audit system. Thus, the forms are checked to see that they have been marked correctly. Accuracy of form-filling is always a problem, particularly with newcomers to the department. It is our practice to approach members individually to point out their mistakes and to return poorly filled forms for correction by the individuals concerned. Feedback of this type reduces the error rate dramatically. We check to see whether all scheduled lists have taken place, and that the audit forms along with a copy of the theatre list reach the anaesthetic office. A cross-check of the number of audit forms against the number of cases on the theatre lists is also performed. Compliance is virtually 100% as a percentage of patients anaesthetised. Regular checks are made to ensure that blank forms are available at every site. The efficiency of our data capture method means that the bulk of the secretary's time is spent in maintaining the office procedures outlined above rather than at the keyboard typing in data.

Workload of the department. A department runs efficiently only if accurate figures about its workload are available. Figure 1 shows the overall workload of our department at the two acute Units over a 9-month period. Eighty-four percent of our theatre work was elective and 16% emergency in nature. A breakdown of the hourly pattern of number of patients anaesthetised in one of the hospitals is shown in Figure 2. The spread of elective workload spilling into unsocial hours is disturbing. There is a wide distribution of emergency work throughout the 24 hours but it would be desirable to move as much emergency operating into the normal working day as possible in line with the recommendations of the confidential enquiry

into peri-operative deaths (CEPOD).⁴ Night-time work is slow, inefficient, expensive and often not in the best interest of the patient. Life-threatening emergencies which require immediate surgical intervention are relatively infrequent and in many instances urgent cases may be postponed safely until the morning without any detriment to the patient. A fully staffed emergency theatre would be required during normal working hours to facilitate this. This would of course mean changes in working practices of surgeons and nursing staff as well as of members of our Division.

Figure 3 shows the number of patients anaesthetised at various sites and reflects the District-wide spread of our practice. Any decisions concerning the anaesthetic department must take into account the diverse number of specialties we serve and the geographical spread of our commitment. A reduction in the number of sites would certainly improve our efficiency.

Teaching and training. Our audit system allows us to monitor the training of junior staff efficiently and accurately. Figure 4 shows the degree of supervision that one trainee received over a period of time. We are able to monitor both the extent of his (her) supervision and the grade of anaesthetists supervising him (her), and can produce a comprehensive report on individual trainees giving details about their theatre activity. Figure 5 illustrates an example of this information. The trainees have found the reports useful (they can supplement a trainee log book) and have welcomed them with enthusiasm. The data collection is so efficient that the figures produced are accurate and up-to-date and do not have to be derived from other observations, such as rotas, which do not necessarily reflect the true picture.

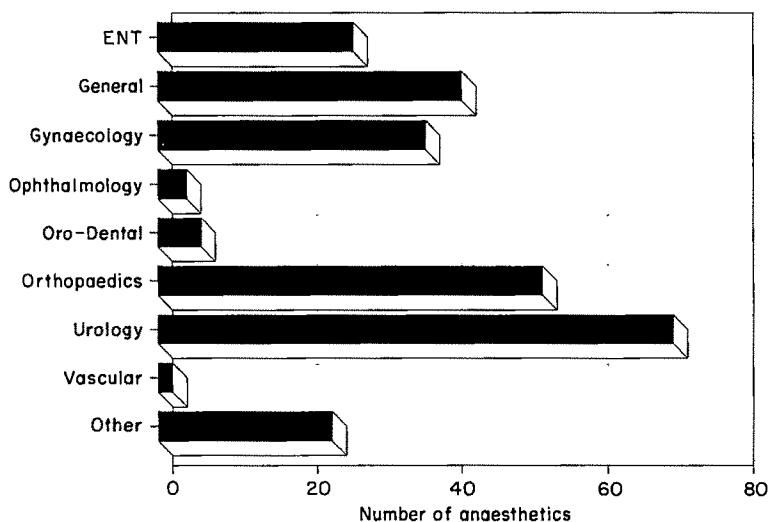


Fig. 5. Experience by specialty.

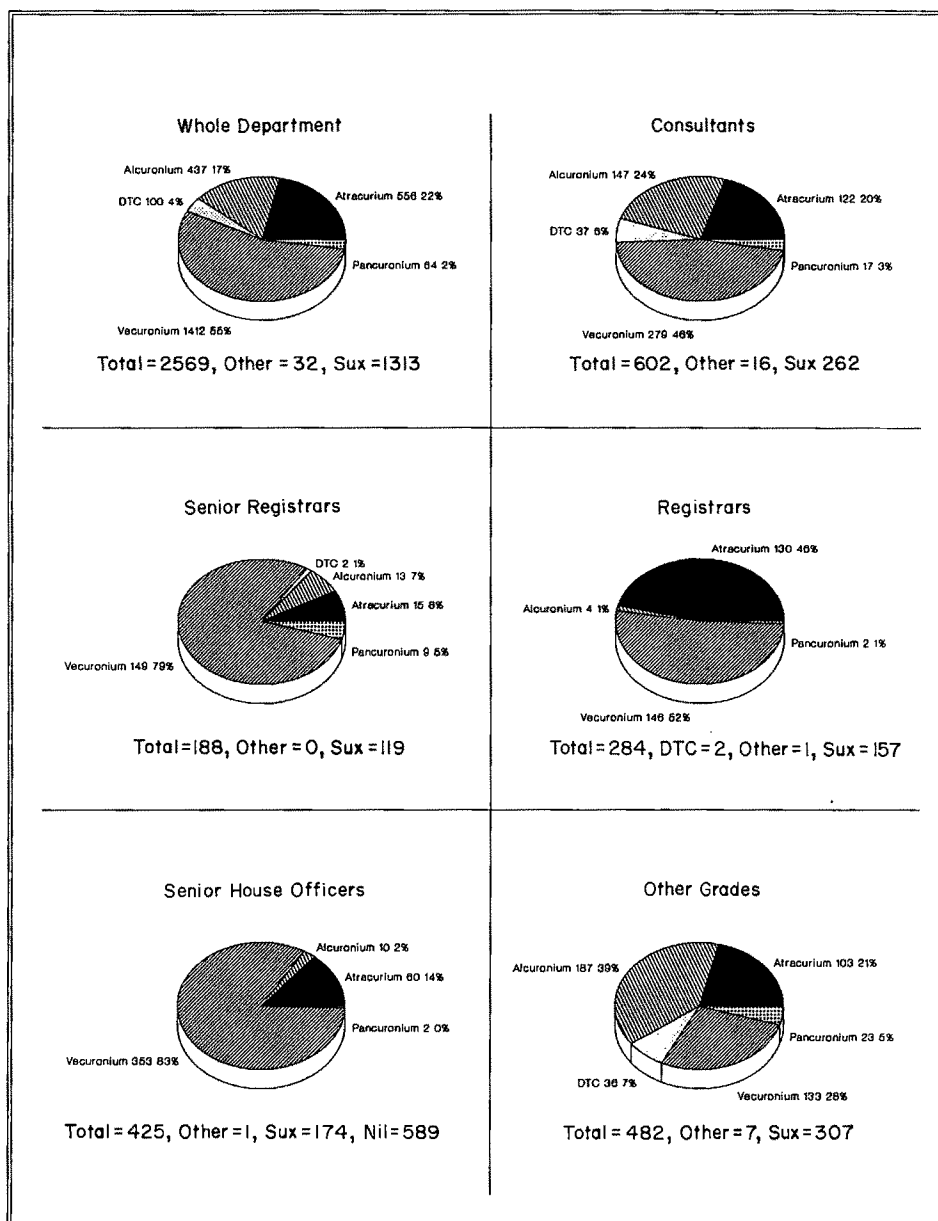


Fig. 6. Derby anaesthetic audit (June–September 1989).

Clinical practice. Our audit system allows us to examine clinical aspects of our work. We record intra-operative morbidity on the back of the audit forms. Forms with positive morbidity are filed separately and used at morbidity and mortality meetings. At one audit meeting we looked at the pattern of drug utilisation by anaesthetists. Figure 6 shows the pattern of use of muscle relaxants by the department as a whole and by individual grades. It can be seen that the senior house officers and registrars tend to use a limited number of the available drugs, although the reasons for this are not yet clear. We would like to encourage them to try to gain experience in the use of some of the other agents available to broaden their experience.

Conclusion

We believe that this audit system is an asset both to the anaesthetic department and the Health Authority. It has been an invaluable tool in monitoring the teaching and

training of junior anaesthetists. On the basis of our initial data we are working with management and our surgical colleagues in planning a more comprehensive anaesthetic service across the District. We are at last beginning to find out about the distribution and patterns of workload which our department undertakes and are starting to look at our clinical practice more critically.

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Anaesthesia for cataract surgery — time for change

The editorial by Dr Rubin (*Anaesthesia* 1990; 45: 717–8) implies that in the USA cataract surgery is performed under local anaesthesia on an outpatient basis since medical insurance carriers will not cover inpatient cataract surgery under general anaesthesia. This is not the case in Calgary, Alberta, Canada, but as in the USA, almost all cataract operations are performed on outpatients under local anaesthesia. The reasons for this are of interest as our National Health Service changes its activities to be more cost-conscious and competitive.

A number of factors came together in Calgary over the past decade to prompt a move from general anaesthesia on inpatients to local anaesthesia on outpatients: the population has aged, so the number of patients requiring cataract surgery has increased with a lengthening waiting list; ultra-violet light exposure because of altitude has increased the incidence of cataract in southern Alberta; surgical techniques for cataract extraction has changed and the use of the phaco-emulsifier and implantation of artificial lenses are now routine; the operating time for cataract surgery has fallen dramatically from a couple of hours to some 20 minutes. As hospital budgets were cut, and Canada has been no different from the United Kingdom in this respect, the move from inpatient to outpatient surgical activities was inevitable, and ophthalmic surgery a good candidate for such management. Indeed, in some centres, cataract surgery is now carried out in free-standing outpatient clinics with no immediate attachment to a hospital.

It is of interest how, over the last 10 years, anaesthetists became involved in providing local anaesthesia for cataract surgery. Initially, the ophthalmologists carried out their own blocks using various sedative cocktail mixtures. This led to frequent calls to the ophthalmic operating theatre to resuscitate patients who had become unconscious, developed respiratory difficulties or were too restless to allow the operation to proceed. Secondly, with the advent of the phaco-emulsifier and the necessity after insertion of the local block to provide external pressure on the eye to reduce intra-ocular pressure, there was considerable delay between cases. This led to very prolonged operating schedules. As a result we negotiated with the ophthalmologists to be taught how to do the blocks, stopped all sedative drugs and overlapped the patients so that surgery could proceed on one patient while ocular pressure was applied to the next in a suitable waiting area. Dr Rubin is correct that operating on an awake, conscious patient requires a re-alignment of surgical thinking, but once this is accomplished all types of patients can be managed. The only patients who are not suitable for local anaesthesia are the very few who refuse and the very young, usually with traumatic cataracts, who cannot cooperate.

The North American experience in many centres show that this elderly, often unwell, population of patients can be safely managed on an outpatient/local anaesthesia basis. Some centres do not insist on pre-operative starvation, do not use an intravenous line and only monitor by pulse oximeter. Some even provide videos of your operation to show to your friends afterwards! By contrast, one of us last year visited a small hospital outside Nairobi, Kenya, and saw cataract surgery being performed on 30 patients per day. The local blocks were administered by a nurse and surgery was carried out by two ophthalmic surgeons. The patients lined up outside the small building used as an operating theatre, had their block inserted, had their surgery performed and went home. The NHS should aim somewhere between these two extremes. Indeed, a start has already been made by using an hotel for immediate postoperative management rather than sending patients straight home.¹

There seems no reason therefore why any cataract surgery should be performed under general anaesthesia in an inpatient setting in the United Kingdom. Anaesthetists should be taught to do the blocks by both the retrobulbar and peribulbar techniques.² The surgeons should be encouraged to accept the benefits of this arrangement and concentrate on their surgical activities.

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L. STRUNIN
P. LEWIS

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In his editorial urging the use of local anaesthesia in cataract surgery (*Anaesthesia* 1990; 45: 717–8) Dr Rubin failed to mention what we believe to be the most important factor in the success of the surgery, namely the patient's willingness to be awake during microsurgery to the eye.

Our Eye Unit uses general anaesthesia for cataract surgery only if local anaesthesia is otherwise contraindicated. The most frequent reason why approximately one third of our cases (1989 figures, 227 of 740 cataract operations) are still being performed using general anaesthesia is because the patient requested it, despite the recommendation of the surgeon and reassurances as to the efficacy of local anaesthesia. No matter how good the akinesia or painless the block, the outcome of the operation is seriously jeopardised by an unwilling and overanxious patient. The American answer to this problem is to administer intravenous sedation or neuroleptanalgesia, which necessitates continuous anaesthetic monitoring and the ability to switch to a general anaesthetic if the patient becomes uncontrollable.

At present, we in the UK can still offer our routine cataract patients a choice of anaesthetic methods and either inpatient or day case surgery. The financial incentives introduced by Resource Management and other health service reforms increasingly pressurise us to reduce this choice.

We believe that safe cataract surgery under local anaesthesia can be done without a full pre-operative general anaesthetic work-up and 6 hours starvation and does not require an anaesthetist in attendance, providing the following criteria are met: the clinically appropriate patient is informed and consents to have local anaesthesia; there is a crash team and full anaesthetic support on site; ophthalmic, anaesthetic and theatre staff know the potential risks and complications of peri and retrobulbar anaesthesia; patients are never converted 'on the table' from local anaesthesia to general anaesthesia. Unsuitable patients (e.g. unexpected claustrophobia) are cancelled and given further counselling before a new date for general anaesthesia is given; intravenous access is secured; no premedication or perioperative intravenous anaesthetic agents are used.

We have established, with the Anaesthetic Department, a protocol for the pre-operative assessment and perioperative monitoring of local anaesthetic cataract patients which is safe and continues to allow us to offer our patients the

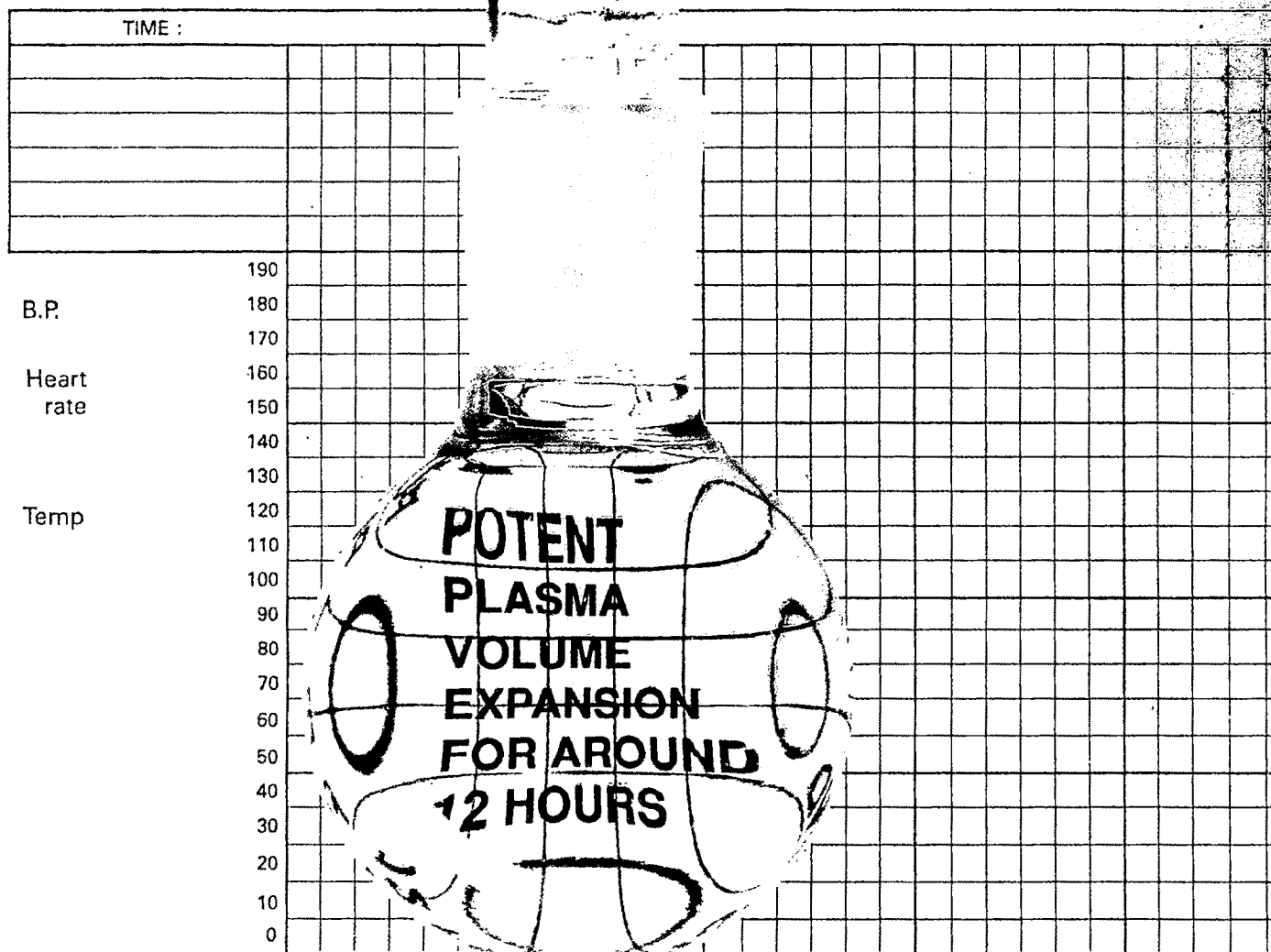
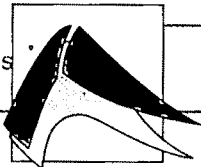
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G.C. VAFIDIS
J.P. BARKER
G.M. HALL

A reply

I thank Professor Strunin and Dr Lewis for their largely supportive comments and respect their experience and knowledge of the subject gained in Calgary and Nairobi. I would just stress that change requires a transition period during which patients, surgeons, anaesthetists and nursing staff must become convinced of the advantages of the proposed alterations. Sudden and complete change is more likely to meet with resistance, the occasional inevitable complication becomes more obtrusive, and there is a risk that the *status quo ante* may be restored irrevocably. For these reasons I believe we should respect the contraindications to local anaesthesia mentioned until much wider experience and greater confidence has been engendered in all the parties involved.

I also agree with Miss Vafidis *et al.* that at the present time about a third of patients still require general anaesthesia. However, I have no doubt that this figure will reduce as patients and attending staff become convinced of the efficacy and advantages of local anaesthesia. Financial incentives must not be allowed to result in a lower standard of patient care or choice.

I believe that the patient requires a full work-up and should be in optimal condition whatever method of anaesthesia is chosen. I am unhappy about a policy to cancel unsuitable patients 'on the table' and 'never to use premedication or peri-operative intravenous anaesthetic agents'. I do not see such a policy as optimal patient care, and consider that a decision to perform an operation should be carried through to the completion of the operation. The presence of a properly trained anaesthetist is likely to result in the correct choice of anaesthetic method, good surgical conditions and allows for the expert and safe use of sedative drugs on the rare occasions they are required.

I would be interested to know the number of the complications of peri- and retrobulbar anaesthetics seen, and how many calls were made of the crash team or anaesthetic staff and with what outcome?

Charing Cross Hospital,
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A.P. RUBIN

We read with great interest the editorial by Dr A.P. Rubin (*Anaesthesia* 1990; 45: 717-9). In the USA, not only cataract surgery but also corneal transplantation, vitrectomy, scleral buckling, strabismus correction and other ophthalmic procedures are done routinely not only under regional anaesthesia but also on an outpatient basis. The vast majority of the adult ophthalmic patients are elderly and can be quite sick, but they tolerate the operations well under regional anaesthesia, and are able to leave the hospital shortly after the end of surgery. It is true that finance played a role in the development of outpatient anaesthesia in the USA, but in our practice we find it is safe to perform ophthalmic procedures in elderly patients under regional anaesthesia on an outpatient basis, and we do recommend it.

As Dr Rubin pointed out, the patients should be cooperative, be able to lie still and fairly flat comfortably and should not have an uncontrolled cough.

University of Texas Medical School,
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Texas
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S.N. KHALIL

Whilst working for Project Orbis, an unusual ophthalmological day care facility based in a DC8 aircraft, I supervised or administered approximately 300 local anaesthetics for ophthalmic surgery. These were a mix of peribulbar and retrobulbar blocks performed on patients as young as 11 years of age and for procedures as complex as penetrating keratoplasty or retinal microsurgery. I must agree with the thrust of Dr Rubin's editorial although I do think that cautious sedation with a benzodiazepine is a useful adjunct to local anaesthesia, particularly in the younger age group.¹ There is no doubt that general anaesthesia is overprescribed for ophthalmic surgery in the United Kingdom.

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J.H. SMITH

Reference

- FRANDSEN J. Local anaesthesia and ophthalmology: Experience with adolescents. *Ophthalmic Surgery* 1989; 20: 680-1.

In his editorial on local anaesthesia for cataract surgery (*Anaesthesia* 1990; 45: 17-8), Dr Rubin states that 'not more than 1% (of patients) will require minimal sedation' for the procedure. He further notes that 'the use of intravenous induction agents to cover the insertion of the block is unnecessary if painless local anaesthesia methods are used'. Our experience on the other side of the ocean, perhaps with more squeamish patients, is quite different. We work in settings where the ophthalmologists prefer to place their own blocks, and which, from what we can observe, are far from painless. Our patients complain bitterly if they are not adequately sedated, especially during the retrobulbar block.

We provide anaesthesia by administering small doses of intravenous midazolam (usually 0.25-1 mg) together with fentanyl (25-50 µg) after monitors are placed. We supply supplemental oxygen via nasal cannula in all patients. We cover the block(s) with minimal doses of methohexitone, usually less than 0.5 mg/kg. After this dose is given, we stabilise the head while the surgeon places the local anaesthetic, meanwhile maintaining the airway and monitoring respiration. Transient apnoea is not uncommon, but saturation by pulse oximetry rarely decreases below the low 90s and apnoea is too short-lived for significant accumulation of CO₂. The patients most often do not move during placement of the block and within 2-3 minutes are awake enough for the surgeon to check motor block. The patients usually lie quietly for the entire operation without requirement of further intravenous sedation. We see very little sudden arousal, and after the methohexitone wears off airway problems are negligible. Afterwards patients are almost uniformly satisfied with their anesthetic care and are able to leave the day surgery unit within an hour.

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P.C. STILLMAN
S. MURAD

A critical incident with the Ohmeda Excel 410 machine

A 75-year-old woman was anaesthetised for repair of a left inguinal hernia. She had a history of hypertension and an hiatus hernia. Her baseline readings before anaesthesia were: arterial blood pressure 125/80 mmHg, pulse rate 90 beats/minute and an oxygen saturation 91% breathing air.

After pre-oxygenation and with the application of cricoid pressure, a sleep dose of thiopentone was administered followed by suxamethonium to facilitate tracheal intubation. She was transferred to the operating theatre and attached to a Manley Blease ventilator supplied by an Ohmeda Excel 410 anaesthetic machine. The gas flows were set with oxygen at 2 litres/minute and nitrous oxide 4 litres/minute with 0.5% isoflurane. Monitoring comprised an ECG, noninvasive blood pressure, pulse oximeter, an inspired oxygen analyser and a capnograph. It was noted that with these flow settings the inspired oxygen analyser showed only 23–25% oxygen and the pulse oximeter an oxygen saturation of 92–94%. The gas flows were changed to 4 litres/minute of oxygen and 4 litres/minute of nitrous oxide; the oxygen analyser then registered 35–36%. A fault was suspected with the analyser so it was changed, but the readings were unaltered. The patient's lungs were ventilated through a Mapleson D system with the same gas flows and the analyser then registered 39–42% oxygen. The ventilator was changed and another brought back into the system but this led to decreasing inspired oxygen concentrations. The capnograph throughout all this time showed an end-tidal carbon dioxide concentration of 5–6.2%, and with gas flows of $O_2:N_2O$ of 2:4 litres/minute it showed an expired nitrous oxide concentration of 80–82%.

The pipelines were disconnected at this stage and cylinder gases supplied with hand ventilation until senior help arrived. The difficulties were demonstrated to a consultant and it was found that with gas flows of $O_2:N_2O$ of 2:4 litres/minute, the inspired oxygen concentrations at both the common gas outlet of the anaesthetic machine and the inspiratory limb of the ventilator were now only 16%. The patient's oxygen saturation decreased to 90% at this stage and the inspired mixture was changed to oxygen 2 litres/minute and air 4 litres/minute, with 1% isoflurane. The analysers now registered 34–36% oxygen and the pulse oximeter showed 93–95% oxygen saturation. It was decided to carry on since the operation was nearly complete and reject the machine afterwards.

The machine was removed from the operating theatre and examined by an Ohmeda service engineer. He found that there was a leak at the top of the oxygen Rotameter which it was believed could be cured with the application of silicon grease, and this was done. However, our equipment unit was not satisfied with this and the machine was removed to their workshop where, the following day in the presence of an official from the Department of Health, the top of the Rotameter block was removed and the 'O' ring at the top of the oxygen flowmeter was found to be split in two (Fig. 1). Thus oxygen was leaking out of the system and an hypoxic mixture was being delivered. The 'O' rings are made of an antistatic fluorocarbon, viton, and are supposed to be changed annually as part of a routine service, but although this machine had been serviced the previous week the rings were not changed. Ohmeda, having discovered this fault, were informed and the other nine similar machines in the operating theatre suite were examined but none had a faulty 'O' ring.

The leak around the 'O' ring was made worse by back pressure from the ventilator and interestingly this particular operation was being performed in the day case operating theatre where the ventilator had not been used



Fig. 1. The Split 'O' ring.

for 5 weeks.

Equipment failure is responsible for only 4% of critical incidents.¹ Holland,² in his review of mortality associated with anaesthesia, stated that hypoxic mixtures as a cause of mortality have disappeared and the problem is largely of historical interest only. In this case a machine, which has only been on sale in the UK since 1987 and purchased by this hospital in June 1988, was found to be delivering an hypoxic mixture. This problem was undoubtedly detected quickly because an inspired oxygen analyser was being used, and such pieces of equipment are now recommended as part of minimal monitoring.

Hammersmith Hospital,
Ducane Road,
London W12 0HS

S. McHALE

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A reply

The fault reported by Hammersmith Hospital to Ohmeda indicated that there had been a problem with the percentage of oxygen being delivered from our Excel anaesthetic machine to the patient via a Manley ventilator. Our service engineer visited the hospital and found a leak had developed at the top of the oxygen flowmeter: the leakage was stopped by tightening the top bobbin stop.

Unfortunately, no Ohmeda representative was invited to be present during the Department of Health inspection and disassembly of the flowmeter. The split 'O' ring was returned to Ohmeda for examination and comment. On receipt by Ohmeda the 'O' ring appeared clean, it was in two pieces and manufactured of the correct material. We are unable to determine when and how it became split.

Ohmeda would endorse the author's comments relating to the use of inspired oxygen monitors and the development of standards for minimum monitoring.

Ohmeda Ltd,
Hatfield, Herts

P. SIM

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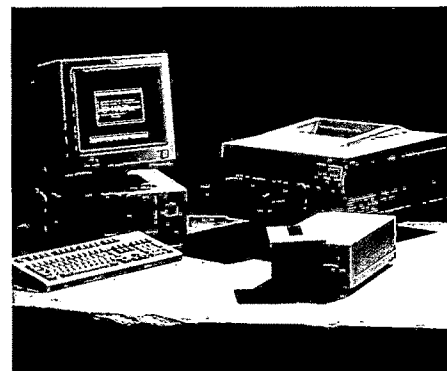


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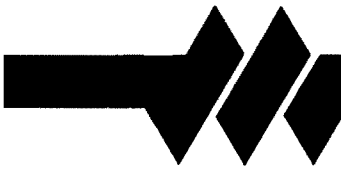


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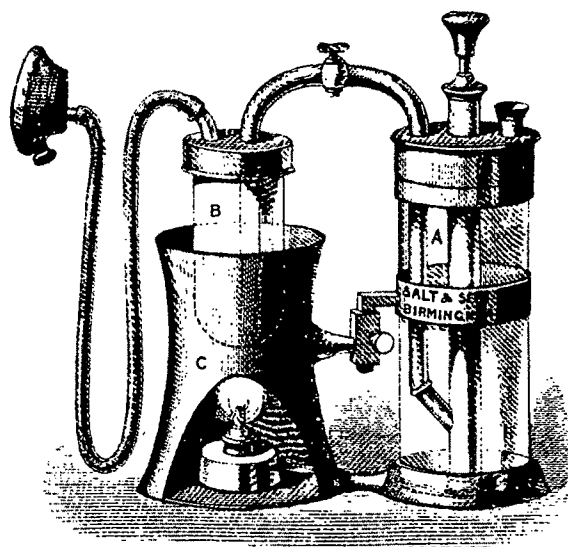
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Atropine or glycopyrronium? — a dangerous dilemma

Glycopyrronium is used increasingly to antagonise the muscarinic effects of neostigmine when neuromuscular blockade requires reversal, because it has a similar pharmacokinetic profile to neostigmine¹ and has no central nervous system side effects.² The total replacement of atropine by glycopyrronium is prevented by the comparative costs of the two agents; 0.6 mg of the latter costs approximately five times more than atropine 1.2 mg (Sheffield District Drug Information Centre). This results in the availability of both agents in the operating theatre with possible confusion because of the large number of different ampoules available. A case is reported where this choice of agents led to a patient developing a serious arrhythmia.

A 66-year-old woman presented for laparotomy with a 2-day history of lower abdominal pain. She had a past history of hypertension, noninsulin dependent diabetes mellitus and depression. These were all well controlled with lisinopril, glibenclamide and clomipramine. Pre-operative investigations revealed a plasma glucose level of 23.2 mmol/litre, urea 9.8 mmol/litre and a WBC of 19.9. All other investigations were normal. Before surgery, a central venous pressure line was inserted and normal saline and an insulin infusion were started. No premedication was given.

Anaesthesia was induced using a rapid sequence technique with etomidate 10 mg, fentanyl 0.3 mg and suxamethonium 100 mg. Neuromuscular blockade was continued with vecuronium 8 mg and the patient's lungs ventilated with 66% nitrous oxide in oxygen and isoflurane 0.5%. The surgery, lasting one hour was uneventful and an inflamed appendix was removed.

Cardiovascular readings remained stable and blood loss was minimal. After reversal of neuromuscular blockade the patient developed ventricular tachycardia which rapidly progressed to ventricular fibrillation. This responded to one DC shock of 200 Joules, but the tachycardia persisted at a rate of 180/minute. The discarded ampoules were examined and it was then realised that the patient had received atropine 1.2 mg (in 1 ml) and glycopyrronium 0.6 mg instead of neostigmine 2.5 mg and one of the anticholinergic agents. Neostigmine 2.5 mg was quickly given and the heart rate settled to 80/minute. The patient was admitted to the intensive therapy unit for observation but developed no further problems and left hospital one week later.

The anaesthetist involved had been on duty for 42 hours at the time of the incident and fatigue was a factor. Confusion arose because of the variable concentrations and mixtures available. This danger could easily be eliminated if only the ready mixed preparations of neostigmine and anticholinergic agents were used for reversal of neuromuscular blockade.

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The supraglottic oropharyngeal airway

We read with interest the letter by Dr Mehta (*Anaesthesia* 1990; 45: 893–4) concerning his experiences with a supraglottic oropharyngeal airway. In it he stated that the average distance from the tip of the tube to the incisor teeth was 10 cm (SD 1.0). We have been evaluating a cuffed oropharyngeal airway, inserted blindly. The airway we used is similar to the one used by Boheimer *et al.*¹ which is now marketed by Portex as a cuffed nasopharyngeal airway (Fig. 1). In our pilot series of 26 patients undergoing minor operative procedures, the cuffed oropharyngeal airway was satisfactory in all but one patient.

However, our results differed from Dr Mehta's study in that we found the measured distance from the tip of the tube to the incisor teeth ranged widely, i.e. between 8.5 to 14 cm. (mean 11.5, SD 1.3) which gives us the impression that the major difficulty with this technique lies in predicting the distance the tube has to be advanced. We found no correlation to either the height or weight of the patient.

We agree with Dr Mehta that this is a suitable method of maintaining the airway. This device is disposable, relatively cheap, and provides a satisfactory alternative to the Brain laryngeal mask; it is particularly useful in our institution which has a high caseload of HIV-positive patients.

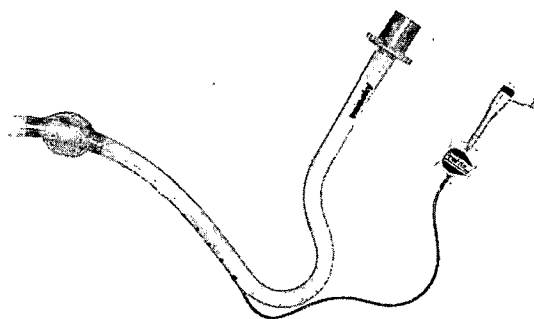


Fig. 1. The supraglottic oropharyngeal airway.

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Reference

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Anaesthetists and trauma

May we comment on Dr Baskett's excellent and timely editorial 'Anaesthetists and trauma' (*Anaesthesia* 1990; 45: 805–7). We can confirm his views on the potential benefits

of the advanced trauma and life support (ATLS) course. Recently a 24 hour on-call trauma team of consultant surgeon, consultant anaesthetist in charge of Intensive

Care, and senior registrar in anaesthetics was set-up, all with an interest in trauma. The team is activated by the casualty officer in the Accident and Emergency department for any patient with a revised trauma score of less than 12 or a Glasgow coma scale of less than 13. All three are ATLS trained and follow the same algorithms and it would seem to have improved our morbidity and mortality figures for major trauma victims (data to be published). Junior members of staff are encouraged to take an active part under the control of the team leader. The same team invariably carried out the operative and postoperative care including intensive care so there is continuity throughout.

We are a relatively small district general hospital (DGH) Accident and Emergency department serving the military

and civilian population, but similar in workload to most DGHs; we believe the ATLS training essential to the efficiency and care of our polytrauma patients.

May we also comment on the wider recognition amongst our surgical colleagues that trauma is not only a surgical orthopaedic disease. The importance of anaesthetic input in the early management of trauma has been found to be invaluable in our practice. After all, up to 90% of traumatised patients do not usually require a surgeon.

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A level vaporizer

Problems with the Ohmeda Enflurtec Mark 4 vaporizer have been reported,¹ when a fluid level was visible in the sight glass although the vaporizer was empty. It was pointed out that awareness during anaesthesia could result from an empty vaporizer that appeared to contain liquid. This false liquid level in the sight glass is caused by liquid sitting on top of a gas bubble, and therefore a few millimetres of liquid can be seen in the sight glass. Apparently this problem has now been overcome by the new British Standard for Anaesthetic and Analgesic machines,² which states that concentration-calibrated vaporizers shall be equipped with a liquid level indicator on which the maximum and minimum levels shall be marked.

However, in our hospital we have noticed considerable tilting of the new, heavy vaporizers on the Selectatec fittings on the back bar. This tilting can lead to a higher level of liquid in the sight glass, and cause the anaesthetist to believe that there is more volatile agent in the vaporizing chamber than there actually is. Tilting can be overcome by a simple modification to the anaesthetic machine: an additional metal supporting bar is fitted below the back bar, onto which two perspex stoppers are attached, just

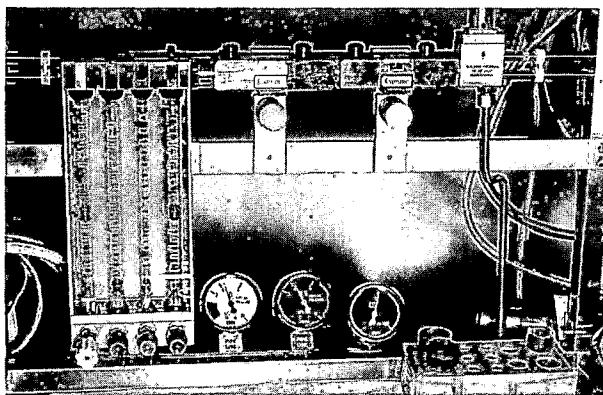


Fig. 1. Position of metal support and perspex stoppers on anaesthetic machine.

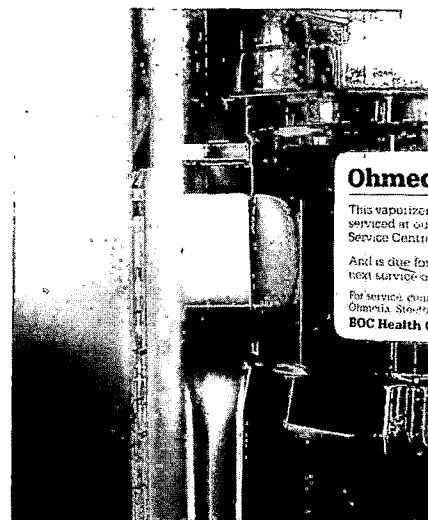


Fig. 2. Close-up view of perspex stopper.

below each vaporizer mounting (Fig. 1). The white perspex stoppers are positioned to support both Mark 3 and Mark 4 vaporizers and maintain them in the horizontal position as shown in Fig. 2. The liquid level in the sight glass now gives a more accurate estimate of the volume of agent in the vaporizer. This modification was carried out by our hospital's Department of Engineering, who service the anaesthetic machines; not only does it stop excessive tilting of the vaporizers, but also alleviates the resultant strain that these vaporizers place on the back bar.

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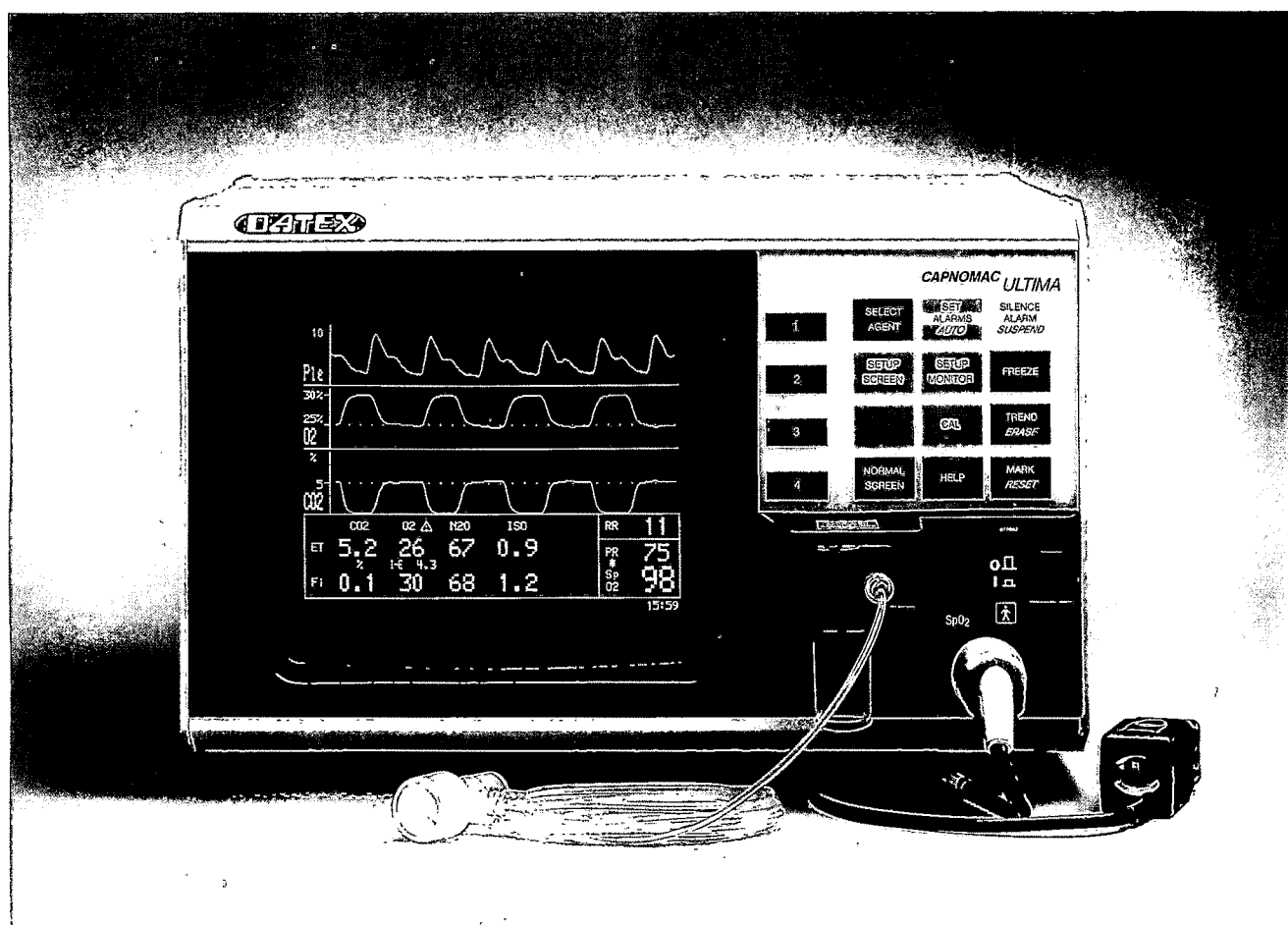
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2. BS 4272. 1989: Part 3; 14.2.

Hazard with a Boyle vaporizer

Recently at our department it was noticed that when using a Manley Pulmovent and Boyle anaesthetic machine in one of the major operating theatres the set minute volume and the expired minute did not correspond. For an average 70-

kg patient the expired minute volume was about 500 to 600 ml less than the minute volume shown on the Rotameter. We were unable to find any obvious leaks from either the system, the back bar or vaporizers, although an

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audible 'hiss' could be heard in the region of the back bar. The Boyle's machine and ventilator were taken out of use and the service personnel contacted. The source of the leak turned out to be from an old Boyle ether vaporizer on the back bar of the machine. The grease around the drum and gas inlet pipe had dried and the drum was worn. When back pressure in the back bar increased during controlled ventilation using the Manley Pulmovent, fresh gas was able to escape between the drum and the gas inlet pipe, exiting to the atmosphere through the slot for the drum-actuating lever. It did not matter whether the actuating lever was in the on or off position. This vaporizer had not been in use for many years and was consequently not serviced,

allowing the grease around the drum to dry out and a significant leak to develop. There are numerous other places in a vaporizer like this where potential leaks can occur, including the bottle sealing washer, the cork stopper or the locking and adjusting rings.

In view of our experience we recommend that this type of vaporizer be removed from the back bar of anaesthetic machines unless it is in regular use and receiving frequent servicing.

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Fixation of tracheal tubes

Fixation of the tracheal tube is a problem encountered by every neonatologist and anaesthetist. Firm fixation is vital,¹ but the present method using gauze strapping or sticky tape on the patient's face is ineffective, unhygienic, and results in potential serious problems. Confused patients and children may pull out the tube and restraining the hands of the patients using tubegauze mittens² is traumatic to them and their attendants. Adhesive tape may damage skin, especially of burns patients and premature babies. Furthermore, a tube firmly fixed with sticky tape, cord ligature, silk suture or cord clamp is difficult to readjust.³ Firm fixation is necessary, and should prevent the tube pulling against the external nares to cause pressure necrosis or kinking, slipping and piston-like movement transmitted from ventilator to patient.⁴

To counter these problems, I have devised a simple and reliable appliance which permits quick fixation and repositioning of the tube (Figs 1-3). This method is hygienic since the device is made of plastic, secretions are easy to clear and conscious patients are at ease. Various combinations of tubes can be fixed using a single device, thus making it easier for the patients to be handled.

The nasotracheal tube fixer has a soft, polypropylene, nose-shaped dome with the tube holder attached to its

anterior border at a slight angle, thereby directing the tracheal tube away from the fragile nasal septum, preventing erosion and pressure on the nares. The lateral edges of the dome have extensions to which strapping is attached, stabilising the holder. These arch around the maxilla and do not apply pressure on the skin of the face. As the material used is very soft, pressure necrosis of the nares and maxilla are avoided.

The oral tracheal tube fixer is a flat plastic sheet with the tube holder attached at its edge on one side. The holder is a C-shaped soft plastic tube with a longitudinal slit on its anterior aspect. The internal diameter of the tube holder is equal to the outer diameter of the tracheal tube. The lock is a firm plastic tube with a graduated bore which when pushed down on to the tube holder causes the longitudinal slit to collapse on the tracheal tube, thus preventing it from slipping. The lock has a small ring internally which fits on to the slit on the outer surface of tube holder. The tube fixer is placed on the upper lip/chin or nose, depending on whether the patient is to be intubated orally or nasally and the strapping adjusted.

When the oral/nasal cavity, visible from anterior aspect, is intubated, the part of the tracheal tube outside the mouth/nose is pushed through the longitudinal slit in the

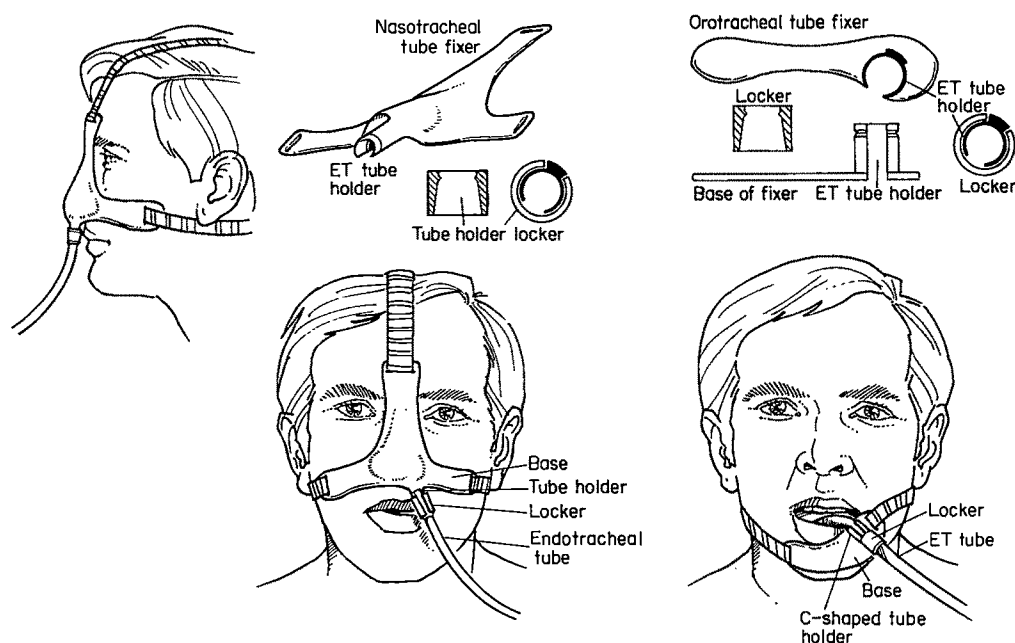


Fig. 1. Diagram of nasal and orotracheal tube fixators.



Fig. 2. Photograph of nasal fixator *in situ* on dummy head.



Fig. 3. Photograph of oral fixator *in situ* on dummy head.

tube holder. Once the tube is in place the ring lock is pushed down on the holder and twisted into the groove provided, firmly holding the tube.

This device would be useful to intensive care personnel, neonatologists, dental surgeons and plastic surgeons since the fixation is reliable and gives better access to the oral and nasal cavity.

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Monitoring of tidal carbon dioxide in spontaneously breathing patients using a mainstream analysing monitor

To the majority of patients anaesthesia is synonymous with pain-free sleep and associated with amnesia. Thus regional anaesthesia frequently requires augmentation with sedatives. Maintenance of a patent airway in a sedated patient is of prime importance and monitoring tidal carbon dioxide will give some measure of adequacy of ventilation, patency of airway and will act as an apnoea alarm. Reliable methods of monitoring $FE'CO_2$ using sidestream analysing monitors have been described for sedated patients^{1–3} and for patients receiving infusions of epidural opioids.⁴ In hospitals and operating theatre suites equipped with mainstream analysing monitors this method of monitoring sedated patients is not possible, but I would like to describe a simple method of monitoring tidal carbon dioxide in sedated patients using a mainstream analysing monitor.

A standard medical suction unit, Ohmeda International, was used to aspirate exhaled gases through the analysing cell where the CO_2 was measured. The suction control unit

was connected through a filter, a 1.6-litre container and a 2-m long tube (ID 7 mm) to a Portex 15-mm female mount. This was attached to the airway adaptor of a Hewlett Packard 14360A CO_2 transducer, mounted onto a 6.0-mm tracheal tube connexion which was connected to an 8-FG suction catheter with terminal and lateral eyes (Fig. 1).

A subatmospheric pressure of 10 kPa is maintained with the suction control at the lowest setting and the unit turned on; this is sufficient to generate a constant flow of 2.8 litres/minute. A satisfactory trace is obtained on the capnograph with the catheter inserted 2–3 cm into the nostril and the transducer placed onto the airway adaptor (Fig. 2). The analysing cell, although a little bulky, can be strapped to the patient's chest or simply laid on the pillow. This adaptation can also be attached to nasal prongs, as suggested Goldman,¹ by using an 18-gauge cannula attached to a 3.5-mm tracheal tube connector mounted onto the airway adaptor. The flow generated with this

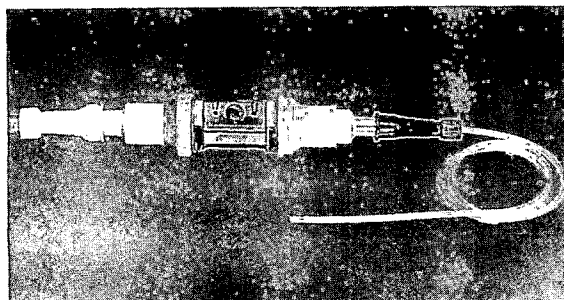


Fig. 1. Hewlett Packard adaptor with suction unit attachment connected to an 8-FG suction catheter.

arrangement is 2.2 litres/minute. There should be no difficulty in modifying this adaptation to fit the Laerdal open mask as suggested by Frankell *et al.*³ and it would be easier than mounting the airway adaptor and transducer directly to the Laerdal mask.

The aspirating flows, although higher than those employed in the sidestream analysing monitors, are the lowest possible to achieve an acceptable tidal CO₂ trace using existing operating theatre equipment, thus avoiding the possibility of aspirating supplementary oxygen. No patients have required alteration of the standard oxygen therapy using a fixed performance oxygen delivery mask

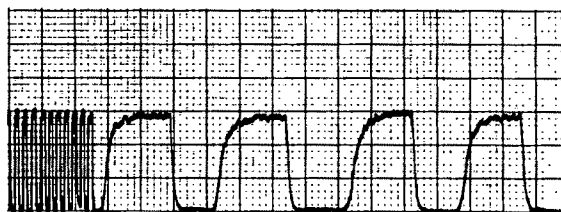


Fig. 2. Capnograph tracing of a spontaneously breathing patient (scale 1 mmHg = 1 mm).

while using this system; all having oxygen saturations greater than 97%.

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Labelling on oxygen analysers

I was interested to read the letter by Dr A. M. Wilson (*Anaesthesia* 1990; **45**: 697) about the labelling of the display on oxygen analysers. Virtually all anaesthesia gas monitoring equipment, including oxygen analysers, capnographs and agent monitors actually measure partial pressures and not percentages. This may cause difficulty in interpretation of the displayed data not only under the circumstances outlined by Dr Wilson but also (perhaps more seriously) where there are significant changes in barometric pressure as may occur at moderate altitude.¹ Similar considerations also apply to vaporizers, the output of which is related to partial pressure and not to percentage. This, too, may lead to misinterpretations and errors where barometric pressures are different from sea level. Dr Wilson's remark that dials or displays on

anaesthetic equipment should be marked with the units actually being measured is to be heartily endorsed. It is surely time that we discarded the time-honoured but scientifically inappropriate percentage value and used partial pressures instead.

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Reference

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Incorrectly filled cylinders

We think your readers will be interested in some bizarre incidents in Hong Kong, where within the past 18 months wrong gases in medical gas cylinders were delivered to two hospitals.

The first occurred in another hospital, when a cylinder supposed to contain oxygen, and labelled as such, was delivered to the hospital and connected to the pipeline to the operating theatres. There, a woman about to undergo a minor orthopaedic operation was given what the anaesthetist thought was a mixture of N₂O and O₂ from the anaesthetic machine. As the patient's condition deteriorated, the N₂O was switched off and what the anaesthetist thought was pure O₂ was given. Cardiac arrest occurred and despite resuscitation, she died on the third post-operative day. It was revealed at the subsequent inquest, widely reported in the local press, that the cylinder in question contained pure nitrogen. No conclusion has to

date been reached as to how the cylinder could have been filled with nitrogen instead of oxygen and how it could have been labelled as oxygen and delivered as such to the hospital.

In the other incidents three patients undergoing emergency Caesarean sections in our hospital in July 1990, were operated on under general anaesthesia. They were given O₂ and N₂O from properly coloured labelled and pin-indexed cylinders on the anaesthetic machine on three different days. They developed diverse signs, delayed recovery being present in all. After the second case, all drugs in the operating suite in the maternity block were changed and the anaesthetic machine, flowmeters and vaporizers thoroughly checked by hospital staff and the Hong Kong Oxygen and Acetylene Company, who found nothing amiss. However, when a third patient displayed adverse signs and symptoms that were otherwise difficult to inter-

pret, the anaesthetists turned their attention to the contents of the nitrous oxide cylinders, which were of the correct colour and pin index coding. It was discovered, using a Cardicap and Capnomac that the cylinder contained carbon dioxide and not nitrous oxide. This was confirmed by the government chemists laboratory. Fortunately all three mothers and their babies recovered fully without suffering long-term effects. Neither the Hong Kong Oxygen and Acetylene Company, the sole manufacturers and suppliers of medical gases in Hong Kong, nor the Royal Hong Kong Police Force, who were looking into these incidents in both hospitals, were able to offer any explanation. The serious

question of how and why the medical gas cylinders were filled with the wrong gases is still unanswered.

This communication should serve as a reminder to all anaesthetists, and others concerned, to exercise utmost care and vigilance at all times and never to take anything, including the contents and/or purity of medical gas cylinders, for granted.

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Potential hazard of central venous cannulation

A 54-year-old patient scheduled for aortic and mitral valve repair for bacterial endocarditis had a triple-lumen catheter inserted via the right subclavian route pre-operatively. Chest X ray on the medical ward confirmed correct placement of the tip of the catheter at the lower half of the superior vena cava (Fig 1). In the anaesthetic room, two 16-gauge single lumen cannulae were inserted via the right internal jugular and the central venous pressure (CVP) was transduced from one of these intra-operatively.

Postoperatively, in the intensive care unit, the central venous pressure wave was transduced via the distal lumen of the subclavian triple-lumen catheter. Initial mean CVP reading was recorded at 18 mmHg. However, the trace was flat and clinically such a high pressure was not anticipated. The line was flushed via the transducer, which was also checked, but with no improvement. Subsequent attempts to aspirate blood from either the middle or distal limbs were not successful, although blood was easily aspirated from

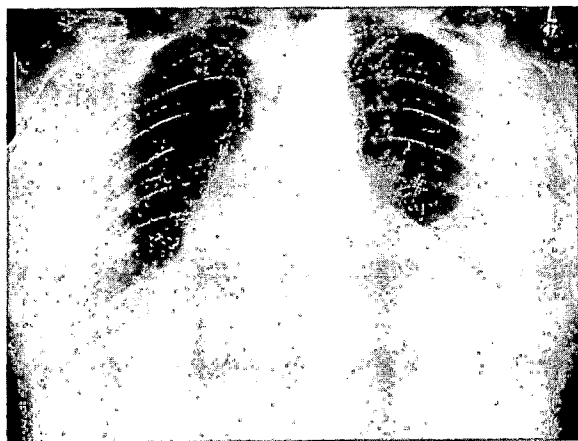


Fig. 1. Pre-operative chest X ray showing tip of triple-lumen catheter in the lower half of the superior vena cava.



Fig. 2. Postoperative chest X ray showing the triple-lumen catheter doubled back on itself, thereby obstructing the two distal lumens.

the proximal lumen and the pressure measured here was 8 mmHg with a more physiological appearance on the trace. Chest X ray revealed that the line had doubled back on itself, kinking and obstructing the two distal luminae (Fig. 2). Review of the operation note provided the explanation. Bicaval cannulae had been inserted for cardiopulmonary bypass and we can only conclude that the superior vena caval cannula had pushed the tip of the central line upwards, thereby bending it.

In the absence of an appropriate pressure waveform technical problems with the catheter should be suspected. This case emphasises that clinical management decisions should be based on a complete assessment of the patient and not upon a single abnormal parameter.

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Intravenous propofol anaesthesia using a computerised infusion system

We would like to reply to the points raised by Dr Goodman concerning our article (*Anaesthesia* 1990; 45: 204-9). We must presume from his statement 'pharmacokinetic parameters that have been derived from groups of patients cannot be applied to individuals because there is too much inter-patient variability' that he has never had the opportunity to use an infusion system such as the one we described. His opinion is not borne out by our own

experience and that of many other anaesthetists who have had the opportunity to use our infusion systems. There are at present 40 similar propofol infusion systems in use and we have obtained detailed feedback from 20 anaesthetists not connected with our own hospital. Eighteen of those claimed that the infusion system has improved their ability to maintain anaesthesia with propofol, principally because they were more confident about the effect of propofol and

that it was easier to use compared with manual control of the infusion.

We agree that the correlation coefficient (r) measures the closeness of the relationship between two variables and not the agreement between them.¹ However, as with any technique, the depth of anaesthesia cannot be precisely predetermined for a given blood concentration of an agent whether predicted or measured. If the predicted blood concentration of propofol is 2 µg/ml and the patient is at a satisfactory level of anaesthesia, then it does not matter if the true concentration is 3.6 µg/ml or 1.1 µg/ml. What does matter is the ability to alter the anaesthetic depth easily and rapidly in a predictable manner. Thus, for example, if the body weight of a patient has been estimated rather than measured, the true blood concentration of propofol may well not be the same as the predicted, but proportional changes in concentration will allow the depth of anaesthesia to be altered in a predictable manner.

While we would agree there may be considerable pharmacokinetic interpatient variability, our experience, and Figure 5 in our article, demonstrate that within-patient variability can be greater than interpatient variability. In practice, the predicted concentration is titrated to achieve the required depth of anaesthesia for any degree of surgical stimulation. Since there can be such a wide variation in the level of surgical stimulation during any operative procedure, the range of predicted concentrations required for

any one patient can vary more than any variation in interpatient pharmacokinetics. We have applied a single set of pharmacokinetic varieties to a widely heterogeneous population of patients and it is to be expected that a certain amount of scatter of data about the line of identity will be obtained in clinical practice. We anticipate that this scatter can be reduced by the use of pharmacokinetic variables which are more finely tuned to individual patients.

We apologise for any confusion arising from the term 'predicted'. This refers to the operation of the real time pharmacokinetic model employed by the delivery system. We did point out that the predicted blood concentration may be different from the true concentration and Figures 2-4 clearly differentiate between measured and predicted concentrations. The term was always used to refer to that concentration which was predicted by the computer, as opposed to the true or measured concentration which existed in the blood at the time of prediction.

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Awareness during Caesarean section

Dr Bogod (*Anaesthesia* 1990; 45: 784-5) states that ventilating the lungs with 50% oxygen during general anaesthesia for Caesarean section establishes fetal hyperoxia and improves the 1-minute Apgar score. The supporting studies are seriously flawed:¹ relief from aortocaval compression was not ensured, the significance of long induction to delivery and uterine incision to delivery intervals was unknown, no attention was paid to maternal carbon dioxide and the control groups and anaesthetic techniques were not standardised.

Two controlled studies using standard anaesthetic techniques and alleviating all currently understood causes of fetal hypoxia show no correlation between maternal inspired oxygen concentrations (of 30% and 50%) and fetal Apgar scores, time to sustained respiration or umbilical oxygenation.^{2,3}

We concur with Dr Lintin that the use of 70% nitrous oxide, in preference to 50%, provides a useful increase in anaesthetic effect as well as increased second gas effect, helping to prevent maternal awareness without detriment to the fetus and allowing a lower concentration of volatile agent to be used.

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Caesarean section. *European Journal of Anaesthesiology*. 1989; 6: 295-301.

A reply

Thank you for the opportunity to reply to the points raised by Drs Matthews, Dann and Cartwright. I agree with them that there are many criticisms to be made of the studies by Marx, Davey, Moir and their colleagues, and I have addressed these elsewhere.¹ The main lesson from these papers is still valid, however; that raising the umbilical vein PO_2 of babies delivered by Caesarean section above that found in normal vaginally delivered babies is associated with a significant improvement in Apgar score. This finding is confirmed by a more recent study.²

The two studies mentioned can both be criticised. Lawes *et al.* did not achieve a significant rise in umbilical vein PO_2 (mean 3.9 kPa) of the babies in their high oxygen group, despite increasing the maternal inspired oxygen concentration from 30% to 50%. Studies which demonstrate an advantage to the neonate of a maternal FIO_2 of 0.5 have always achieved an umbilical vein PO_2 of 4.9-5.2 kPa. The reasons for this failure are unclear, but it is hardly surprising that these babies showed no benefit.

The paper by Dr Matthews and his colleagues is one which concerns me greatly, and I was surprised that no adverse comment was made at the time of publication. In this (fortunately) small study, half of the patients, following thiopentone induction, had their anaesthesia maintained with 50% oxygen and 50% nitrous oxide, with no volatile or opioid supplement until delivery of the baby. The authors themselves admit that this method is associated with an awareness rate of 10-25%, and yet made no special attempt to monitor for awareness during the procedures. Ethical questions aside, the high level of circulating catecholamines in these 'anaesthetised' patients would cause marked uterine artery vasoconstriction, resulting in a

failure to hyperoxygenate the fetus (as demonstrated by mean umbilical vein PO_2 of 3.92 kPa in this group).

In conclusion, Dr Matthews and his colleagues have misinterpreted my hypothesis. It is umbilical vein PO_2 and not maternal inspired oxygen concentration which correlates with neonatal well-being. In most cases, a rise in the former is best achieved by increasing the latter.

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The Engstrom Elsa anaesthetic machine

The assessment of the Engstrom Elsa (*Anaesthesia* 1990; **45**: 746-50) was of particular interest to us. Our department bought three of these machines, equipped with the CO_2 absorber, principally because of economy of gases and vapours, simplicity of use and cleansing, and the incorporated monitoring and alarm systems seemed advantageous. While some of these reasons have been vindicated, numerous problems have been encountered.

The familiarisation period has been fraught for all grades of staff and necessitated some changes in practice, particularly when compared with the circle system and CO_2 absorber of our other anaesthetic machines, the Ohmeda Modulus II plus with Tec 4 vaporizers. The Engstrom

Elsa's fresh gas and inspired vapour concentrations are very slow to respond to alternations in vaporizer settings. Figure 1 shows representative findings for both machines in IPPV mode with a fresh gas flow of 4 litres/minute. A dummy lung was used. Halothane concentrations were measured at the angle piece. Identical patient tubing was used.

Figure 2 shows the response of the vaporizers to changes in settings. Halothane concentrations were measured at the external fresh gas outlet of both machines with a fresh gas flow of 8 litres/minute via a Bain system. A calibrated Datex Capnomac was used to measure halothane concentrations.

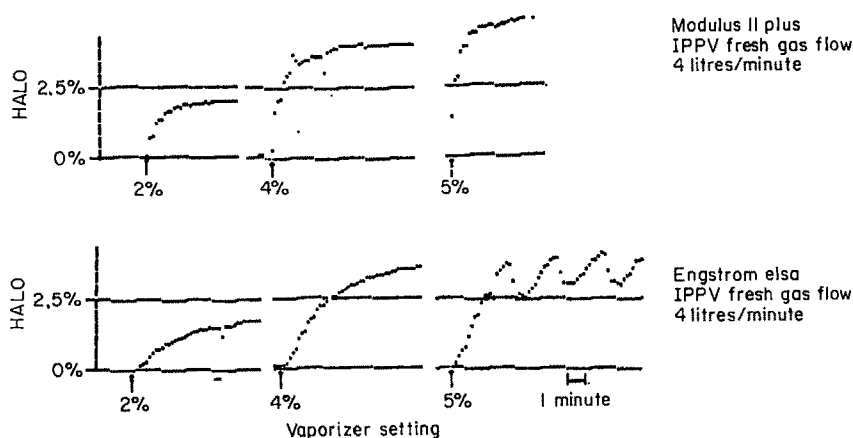


Fig. 1. Inspired halothane concentration during IPPV at vaporizer settings of 2%, 4% and 5% halothane.

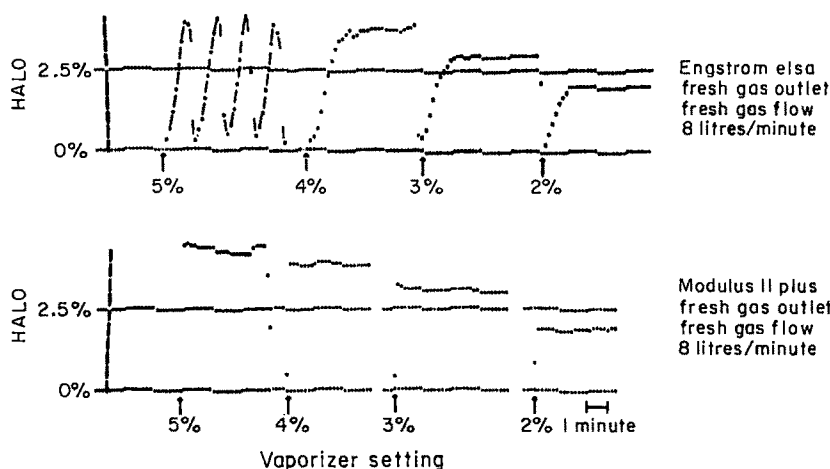


Fig. 2. Response of vaporizers at settings of 2-5% halothane with a Bain system attached to the external fresh gas outlet.

The Engstrom Elsa vaporizer has a maximum dialled setting of 8% and a lock which must be bypassed for settings above 3%. A maximum inspired anaesthetic concentration of 4% is permitted by the Engstrom Elsa and gas sampler (Aridus), irrespective of the volatile anaesthetic agent used. The vaporizer at this point automatically switches off and output decreases before slowly increasing to 4% again. This is illustrated in Figures 1 and 2 for both IPPV and spontaneous ventilation modes using a dialled vaporizer concentration of 5% halothane. The inspired concentration during IPPV fluctuates between around 2.5% and 4%. However, during spontaneous ventilation (induct mode) the inspired concentration fluctuates markedly and a clinically useful inspired concentration is not delivered. Initially, this resulted in problems from light anaesthesia after transfer from the anaesthetic room to the operating theatre Engstrom Elsa. Two patients under mask and airway anaesthesia vomited while supine as a consequence of this.

The problem can be avoided either by attaching another anaesthetic breathing system to the external fresh gas outlet and removing the gas sampler, or by flushing the system with volatile agent between cases. The former should not be necessary and risks delivering 8% inspired vapour inadvertently, and the latter option is wasteful and pollutant.

The patient Y-piece and gas sampler are bulky and awkward to hold. The CO₂ absorber is mounted low down and so tubing tends to 'drag' at the patient connexions. Our machines have also given the occasional assisted breath

during spontaneous ventilation despite an inspiratory assist setting of zero. The manufacturers have been made aware of this.

The manual bag has two functions: manual ventilation or the monitoring of respiratory movements of the patient bag-in-bottle during spontaneous ventilation. According to its position on the lower monitoring rail, the CO₂ absorber can keep the valve of the manual bag open and the manual bag deflated. This prevents both of the above functions. It is very awkward manually to ventilate a patient's lungs when there is even a slight leak around a facemask; the manual and patient bags deflate and operating the separate filling controls for both bags while manually ventilating is cumbersome.

In the event of an electrical power failure the vaporizer output stops. However, when power returns, the vaporizer must first be switched off and then on again before it will deliver any more vapour. The power failure alarm can also be irreversibly silenced for the duration of the power failure.

In practice, the Engstrom Elsa is not well suited to short anaesthetics with spontaneous ventilation where the level of anaesthesia needs to be rapidly deepened. In other respects the machine is simple to use but the familiarisation process is lengthy.

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S.J. ROWBOTTOM
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Scavenging in the operating theatre

Routine checking of the breathing system, as with all anaesthetic equipment, before the start of an operating theatre list is mandatory. Not uncommonly, a coaxial Mapleson D or Bain system is found to be faulty because of a leak in the inner or outer tubing. This means the apparatus has to be discarded.

We describe a potential use for such a system in situations where operating theatre scavenging is insufficient or not available. If connected to the expiratory valve of a Magill breathing system, it can be used as a scavenging device. Clearly, the breathing system must be labelled as being no longer safe for normal use. The air brake, commonly seen in active operating theatre scavenging equipment, uses suction applied to an inner tube with air entrainment in the outer tube. This can be mimicked using the coaxial system; the inner tube attached to a suction

source will provide active scavenging, while the outer tube supplies air entrainment and protects the patient from a high subatmospheric pressure. Several small perforations can be made in the outer tube to enhance air entrainment. We have found that this system works well in the spontaneously breathing patient. The expiratory valve on the Magill attachment functions normally and we have not experienced any problems in its use. Operating theatre pollution is considerably reduced at no extra cost and with little difficulty.

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P. AMOROSO
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Defective spinal needle

Spinal anaesthesia was chosen for a Furlong hemiarthroplasty in a 92-year-old woman with a left subcapital femoral fracture. A 26-gauge Becton Dickinson spinal needle was inserted through its 3-cm 20-gauge introducer needle at the L₃₋₄ interspace and the subarachnoid space was encountered at 6 cm without difficulty. Clear fluid was observed to drip from the hub of the introducer needle at the start of a spinal injection of 0.5% isobaric bupivacaine; this ceased when the injection was stopped. It was obvious that the tip of the introducer needle could not be in the subarachnoid space and the injection was continued to a total volume of 5 ml, during which time 2 ml were estimated to have spilled from the

introducer, giving an estimated intrathecal dose of 3 ml, as had been intended. A successful block resulted and surgery proceeded uneventfully.

Inspection of the spinal needle afterwards revealed a small defect in the midshaft of the needle which permitted the efflux of local anaesthetic (Figs 1-3). This defect was not obvious before insertion. Had the defect not been revealed by the appearance of leaking fluid from the introducer, as would have been the case had an introducer not been used or had the defect occurred nearer the needle tip outside the lumen of the introducer, an inadequate block could have resulted with normal dosage. However, the possibility of breakage or fracture of the needle at its

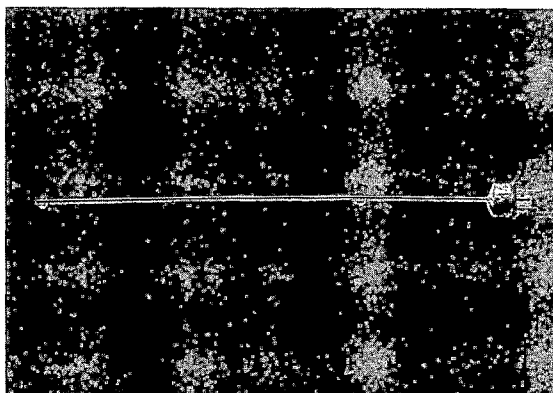


Fig. 1. Local anaesthetic emanating from distal needle hole and midshaft defect.

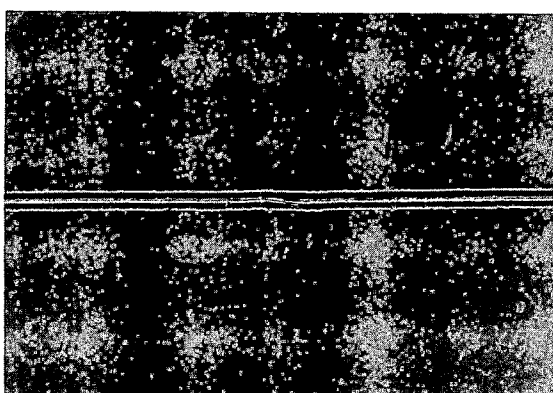


Fig. 2. Magnified view of midshaft defect.

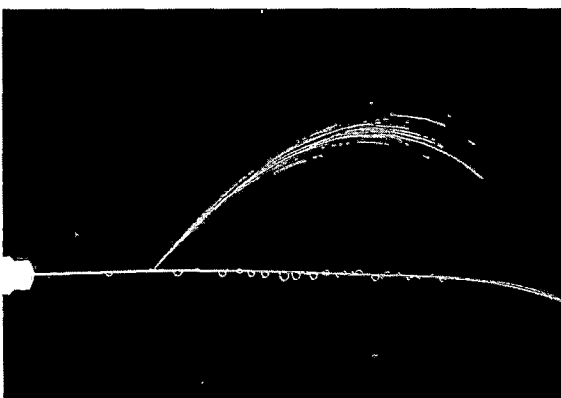


Fig. 3. Local anaesthetic injected under pressure escaping from the distal end of the needle and from the defect.

weak point within the patient would have been of greater concern.

Anaesthetists are responsible for checking their equipment, but it is possibly a counsel of perfection to recommend minute inspection of every disposable needle used. Most practitioners rely on the quality control of the manufacturers, which, as is demonstrated in this case, is not always infallible.

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A reply

Thank you for giving us the opportunity to reply to the letter above which outlines a failure in use of one of our spinal needles. Tests were undertaken on the spinal needle and the Becton Dickinson history files were reviewed for the period from 1981 to the present for any complaints regarding broken/split spinal needles.

This defect was caused by a small particle causing a nick or gouge to the cannula surface before the cannula was drawn down into its current gauge. This spinal needle cannula is produced by pulling down a tube of metal through a series of dies until it is the appropriate needle diameter. If a small surface nick or gouge occurs early in the pull down it will enlarge and deepen through this process. Obviously, as the tubing diameter becomes smaller in size the wall thickness thins simultaneously and as a 26 G spinal needle has extremely thin walls a nick is more likely to form a split or crack.

We visually inspect all cannulae at three separate stages at our cannula manufacturing and assembly plants to prevent defects like this from going to market. With the increased use of smaller gauge needles, Becton Dickinson has realised the difficulties of visual inspection and is therefore introducing an automated system which will reject any such cannula and stop this fault occurring again.

A review of our files shows that this complaint is the only one of its kind we have had in the last 10 years. During the last decade, we have manufactured over 100 million spinal needles, so as you can see this type of complaint is extremely rare.

Medical Division,
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G. STEVENSON

Measurement and manipulation of pulmonary artery wedge pressure in adult respiratory distress syndrome

As part of a larger study of adult respiratory distress syndrome (ARDS) we conducted a postal survey of 299 British intensive care units to determine firstly, practice in the measurement and manipulation of the pulmonary artery wedge pressure (PAWP) in patients with ARDS, secondly, methods employed for doing so and thirdly, methods used to monitor adequacy of oxygenation. The Directory of Emergency and Special Care Units 1989 (CMA Medical Data Ltd, Cambridge) was used as source material and questionnaires were sent to all units using the word 'intensive' in their title. We did not follow up nonresponders.

One hundred and sixty-seven replies (56%) were received and 132 ICUs (79%) use pulmonary artery catheters. In response to the question 'what value of PAWP do you take as the cut-off between high and low pressure pulmonary oedema?', the mean (SD) quoted 'watershed' value was 16.1 (2.8) mmHg. Respondents were asked to define an optimal PAWP in a patient with ARDS and normal left ventricular function; the mean (SD) suggested optimal PAWP was 10.6 (4.4) mmHg (Fig. 1). Results concerning the methodology of measurement of PAWP are displayed in Table 1.

Ninety-eight respondents (74%) claimed to reduce the

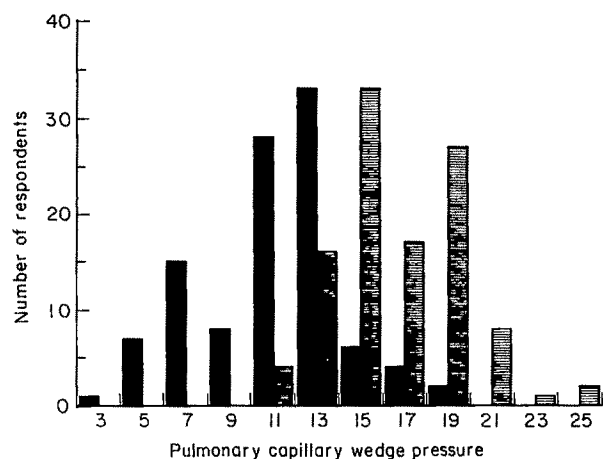


Fig. 1. Optimal PAWP in a patient with ARDS and normal left ventricular function. ■, optimum pressure; ▨, watershed pressure.

PAWP in ARDS deliberately, 32 respondents (24%) do not do so and two did not answer this question. Methods employed to reduce PAWP included diuretics (89 (91%) respondents), fluid restriction (88 (90%) respondents), haemofiltration/haemodialysis (53 (54%) respondents), venodilators (70 (58%) respondents) and several respondents mentioned the use of inotropes.

Not all completed the section on monitoring adequacy of oxygenation. P_{aO_2} is used by all 149 respondents to this question, S_{aO_2} or S_{pO_2} by 115 respondents (86%), $\bar{S}v_{O_2}$ by 54 (51%), $\dot{V}O_2$ and $\dot{D}O_2$ by 59 (55%); the use of $\bar{S}v_{O_2}$ featured in 38 replies (38%), cardiac output in 89 (74%), lactate concentrations in 27 (26%); nine respondents (7%) claimed not to use pH.

Table 1. Methodology of measurement of PAWP.

	Number of ICUs (%)		
	Yes	No	No answer
Removal of PEEP	43 (33)	66 (50)	23 (17)
Stop ventilation	27 (20)	82 (62)	24 (18)
Measurement at end expiration	70 (53)	35 (27)	27 (20)
Measurement without altering ventilation	64 (48)	35 (27)	33 (25)

Diagnostic fiberoptic bronchoscopy using the laryngeal mask

General anaesthesia for diagnostic fiberoptic bronchoscopy usually involves tracheal intubation which then obscures the larynx and upper trachea, which may be sites of pathology. The trauma of tracheal intubation may also disrupt pathology in the latter site. The laryngeal mask airway (LMA) provides an alternative method in which vocal cord movement can be observed and the upper trachea inspected and biopsied unhurriedly.

After induction of anaesthesia and insertion of the LMA the fiberoptic bronchoscope can be used to ensure correct positioning of the LMA and observe cord movement during spontaneous ventilation. Our preference is to then paralyse and ventilate the patient's lungs to prevent coughing as the bronchoscope passes between the vocal cords and because recovery from anaesthesia is quicker. It

The moderate response rate of 56% is typical of postal surveys,¹ but does represent at least a substantial body of opinion in British intensive care. Previous surveys have concentrated on usage rates, complications and outcome of pulmonary artery catheters rather than the philosophy behind the manipulation of PAWP.² We noted a large variation of opinion on both the optimal and 'watershed' pressures and there was considerable overlap (Fig. 1). The mean (SD) difference between optimal and 'watershed' pressures in 89 replies was 5.9 (3) mmHg.

The validity of the use of PAWP as a measure of true pulmonary capillary pressure has been questioned^{3,4} but it remains common clinical practice. Although the prevention of a rise in PAWP seems logical to prevent an increase in lung water, there is more contention in the deliberate reduction in PAWP in ARDS. There is little evidence in the literature that reducing PAWP (from an already normal or low value, by definition in ARDS) reduces lung water, shunt or increases P_{aO_2} , which are the expected outcome and hence advantageous. If the expected outcome is not of proven benefit, the reduction of PAWP certainly does reduce cardiac output and hence $\dot{D}O_2$ at a time of great risk of multiple organ failure, and some authors have shown increased survival in patients with augmented $\dot{D}O_2$ and $\dot{V}O_2$.⁵ Is it possible that the potential reduction in cardiac output, $\dot{D}O_2$ and $\dot{V}O_2$ (unless supported by increased inotropes) from a reduction in PAWP to 'subnormal' levels is a greater cost than the potential and unproven benefits of a reduction in lung water?

Further research to define the precise effects of PAWP reduction in ARDS would be of great benefit to the critically ill patient.

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should be stressed that to avoid gastric inflation, gentle (manual) ventilation with a Bain system is used. Appropriate modification of the angle piece (15 mm Portex swivel connector) enables the bronchoscope to be passed down the lumen of the LMA while delivering anaesthetic. We have also used this method to observe vocal cord movement after thyroid surgery.

In adults an Olympus BF 2TR fiberoptic bronchoscope passes through the grille of a size 3 or 4 LMA without undue friction and in infants and children an Olympus ENF P2 passes easily through size 1 and 2 LMAs.

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A new way to measure the width of the epidural space

Nickalls and Kokkri (*Anaesthesia* 1986; 41: 432-3) measured the width of the epidural space by introducing a 26-gauge spinal needle through a 16-gauge Tuohy epidural needle. The spinal needle was clamped with an artery clip as soon as the dura was felt to have been pierced. There is some inaccuracy in their measurement because the tip of the spinal needle is bent when it passes through the tip of the epidural needle, so it does not cross the epidural space in the shortest distance from the ligamentum flavum to the dura.

To overcome this inaccuracy, we developed a way in which the width of the space can be measured using the obturator of the epidural needle (Fig. 1). The latter is advanced until the epidural space is reached. A spinal needle is then inserted in an adjacent interspace and advanced until CSF is obtained. The point where the spinal needle pierces the skin is marked with a small artery clip (point E). This is the distance length from the skin to the point where the spinal needle pierces the dura (length L). The length of the epidural needle from the skin to the epidural space is measured on the epidural obturator (length d), which is equivalent to the inside length of the epidural needle. By subtracting length d from length L the width of the epidural space is obtained, providing of course that neither the spinal nor the epidural needles are pushed further into the epidural or intrathecal spaces. In this way

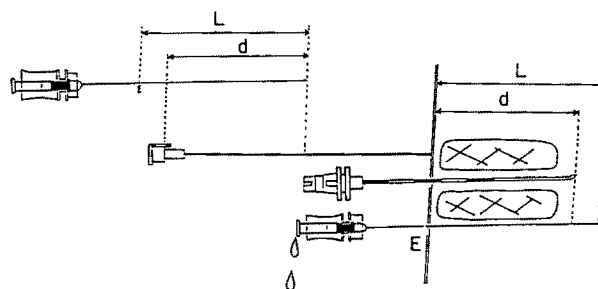


Fig. 1. By subtracting length d of the epidural obturator from length L of the spinal needle the width of the epidural space is obtained.

the width of the epidural space can be obtained more accurately. The importance of this width is increasing as combined spinal-epidural anaesthesia becomes more popular.

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Amorous behaviour and sexual fantasy after anaesthesia or sedation

Doctors Boheimer and Thomas (*Anaesthesia* 1990; 45: 699) quoted Bricker (*Anaesthesia* 1988; 43: 171), and stated that amorous or disinhibited behaviour occurred in more than 12% of 130 anaesthetics in which propofol and alfentanil were used in minor gynaecological procedures. They suggest that susceptible patients should be forewarned of this possibility, in an attempt to prevent postoperative embarrassment, should recall occur. How do they identify susceptible patients? Which subgroup of the population are more likely to react in this manner? If we cannot accurately predict those patients, do we forewarn everyone? If so, we potentially embarrass 100% of our

patients on the basis that 12% may exhibit this behaviour, and that an unknown fraction will have recall. How should anaesthetists on their pre-operative visit explain to a 15-year-old girl, in front of her parents, that sexual fantasy and amorous behaviour might occur? I agree that a third party should be present, for medicolegal reasons. However, I believe that it is neither practical nor desirable to forewarn patients of this potential side effect.

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Elohes administration following an anaphylactoid reaction to Haemaccel

A 6-year-old boy weighing 20 kg presented for craniotomy and excision of recurrent cerebral tumour. The patient had a history of eczema and asthma and was sensitive to thiopentone, suxamethonium and alcuronium. He suffered a life-threatening anaphylactoid reaction to Haemaccel 3 weeks before. The patient was premedicated with atropine 0.5 mg and received nebulised salbutamol one hour before operation. Anaesthesia was induced with cyclopropane in oxygen, vecuronium 2 mg and fentanyl 100 µg. The trachea was intubated and the lungs ventilated to an end-tidal carbon dioxide concentration of 3.5 to 4% with 33% oxygen, 66% nitrous oxide and 1% isoflurane. Elohes (20 ml/kg) was infused to replace blood loss during surgery. No adverse reaction occurred during the conduct of anaesthesia.

Elohes is a 6% colloidal solution of hydroxyethyl starch (median molecular weight 200 000) in physiological saline. It has an osmolality of 322 mOsm/kg H₂O and is non-antigenic. Intravenous infusion results in immediate expansion of plasma volume in excess of the volume

infused. The clearance rate from blood is rapid and more than 40% of the total dose appears in the urine within 24 hours.

Haemaccel, a urea-linked gelatin, is known to produce anaphylactoid reactions as a result of histamine release and could be considered an inappropriate colloid to use for such atopic patients. The direct histamine releasing effect of urea-linked gelatins is due to an excess of di-isocyanate. The amount of free di-isocyanate has now been reduced by improved purification resulting in a reduced frequency of anaphylactoid reactions,¹ but fatalities are still reported.² In contrast, the administration of Elohes is not associated with raised plasma histamine levels. Furthermore, the molecular weight distribution is such that complement activation is also less likely. We would advocate Elohes as a suitable plasma expander for use in atopic individuals.

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Epidural analgesia and hypotension

Drs Shorten and Furness (*Anaesthesia* 1990; **45**: 169) described severe hypotension caused by bladder distension in a 72-year-old diabetic man with autonomic dysfunction. The patient had had a crossover femoropopliteal graft inserted under spinal anaesthesia. A recent experience suggests this problem may not be confined to elderly diabetics.

A 28-year-old primiparous patient in labour requested an epidural for pain relief. Her circulation was preloaded with 500 ml compound sodium lactate solution, and an epidural catheter was inserted in the L_{2-3} interspace. A test dose of 3 ml 0.5% bupivacaine was followed by 10 ml of 0.25% bupivacaine 10 minutes later, which produced complete analgesia. A second 50 ml bag of compound sodium lactate solution was started, to run over 6 hours and an infusion of syntocinon was also commenced. Twenty minutes after the epidural was inserted an infusion of 0.25% bupivacaine was started at a rate of 5 ml/hour. Her arterial blood pressure was monitored every 5 minutes for the first half hour and quarter hourly after that. One and a quarter hours after the epidural was inserted her blood pressure was found to have decreased from 100/60 mmHg to 75/50 mmHg. She was lying on her left side, the epidural block was at the level of T_{10} bilaterally with minimal motor blockade, and it was still providing complete analgesia. She was given a further 500 ml of compound sodium lactate solution and a total of 30 mg of

ephedrine in 6-mg increments, which increased her blood pressure to 95/50 mmHg.

Further examination revealed her to have a distended bladder; a catheter was inserted and 1100 ml of urine was drained. Following this her arterial pressure increased to 115/60 mmHg. She remained stable over the next 4 hours until her blood pressure was again found to be decreasing, initially to 90/60 mmHg, then to 80/60 mmHg. Again she was on her side with a block to T_{10} . A urinary catheter was inserted and 600 ml urine drained. Her blood pressure increased to 105/60 mmHg without any other treatment. A live baby boy was delivered 2.5 hours later.

The rapid resolution of the hypotension on both occasions following drainage of the bladder implicates bladder distension as a major contributory factor. The degree of hypotension and its resistance to treatment was not in keeping with the level of the epidural blockade. In conclusion, drawing on this experience and the experience of Shorten *et al.*, bladder distension can cause significant hypotension in patients undergoing both spinal and epidural anaesthesia, and this easily remediable cause should be borne in mind particularly when the degree of hypotension is not in keeping with the level of the block.

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Use of the laryngeal mask airway in primary care

We would like to confirm Major De Mello's impression that the laryngeal mask airway (LMA) can be inserted easily by paramedical personnel (*Anaesthesia* 1990; **45**: 793–4). In a controlled trial, we compared laryngeal mask insertion with tracheal intubation in anaesthetised patients.¹ Eleven naval medical ratings performed a total of 110 sequential insertions. The overall success rates were 51% and 94% for tracheal intubation and LMA insertion respectively ($p < 0.01$). In addition, the LMA was inserted significantly quicker ($p < 0.01$).

We agree that the LMA may have a place in resuscitation but are anxious to point out that further controlled studies should be conducted to establish the advantage or otherwise of the LMA over bag and mask techniques, the actual risk of aspiration using the LMA

and the value of the LMA in full cardiopulmonary resuscitation. Until these studies have been completed, use of the LMA is still contraindicated in primary care.

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Reference

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Visual disturbances after transurethral resection of the prostate

It was interesting to read Dr J. N. Cashman's letter in your recent correspondence column (*Anaesthesia* 1990; **45**: 691). I disagree with Dr Cashman's view that transient blindness was due to glycine-induced oedema of the visual cortex. Although the author has not mentioned the 'blink and menace' reflexes, the presence of perception of light and poor light reflex is contrary to the diagnosis of cortical

blindness.¹ Also, how can one explain the occurrence of glycine-induced oedema in only the occipital lobe? I wonder whether transient blindness might have improved spontaneously without diuretic therapy, as seen in one of the cases reported by Andranik *et al.*²

It seems likely that glycine, being an inhibitory neurotransmitter, and its level being high in retina, might

produce transient blindness due to retinal dysfunction. A visual evoked response may be useful in such cases.

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Obituary — Dr G.S.A. Knowles

May I correct the obituary notice (*Anaesthesia* 1990; 45: 905) relating to Dr G.S.A. Knowles, whose death I was very sorry to read about. He qualified at Leeds in 1939, and was for some 25 years the senior anaesthetist at the North Middlesex Hospital, London. He was one of the small group who met in March 1954 to found the North London Group of Anaesthetists and was its first chairman. It was at one of the early meetings of the Group that he acquainted us with the Knowles classification of prostatectomy patients at the North Middlesex:

1. unfit; 2. very unfit; 3. grossly unfit; 4. extremely unfit; 5. poor risk. Knowles was a master of all techniques, including one-upmanship, and a great exponent of the art of spinal anaesthesia. During the last few years he lived in retirement in Suffolk.

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D. ZUCK

Foreign body in a laryngeal mask airway

During a routine operation for transurethral resection of prostate I was alerted to the unusual form of respiratory monitoring that suddenly had become apparent. The patient's airway was maintained with a re-autoclaved laryngeal mask airway (LMA). He was breathing comfortably, spontaneously, through a circle system with in-line soda lime absorber. Catheter mount oxygen level and end-tidal carbon dioxide were at 30% and 5% respectively. Pulse oximetry registered 94% saturation. Pulse rate and arterial blood pressure were normal. There was nothing untoward other than a 'bobbin' moving up and down inside the visible length of the LMA in time with the phases of respiration. Removal of the catheter mount allowed this foreign body to be launched to atmosphere.

It proved to be a one-way return valve used normally to seal the cuff on an LMA. Its passage into the patient's trachea had been prevented by the grill at the distal end of the LMA. It was surmised that it had become sequestered at some time during the cleaning process, and concealed by being fixed by autoclaving in the 'blind' spot formed by the

junction between the opaque rubber and transparent plastic that constitute an LMA. The recommended preparation of an LMA immediately before insertion emphasises the placement of the 'business end' on a flat surface to empty the cuff. It is then lubricated and inserted by the anaesthetist with the laryngeal surface out of the line of sight of the inserter. The presence of the foreign body might have been spotted had the LMA been examined upside down beforehand.

Far be it for this experience to be seminal in a literature of foreign body exotica in LMAs. Perhaps the reporting of this potential catastrophe and advocacy of the simple expedient of examining the equipment's blind spot will prevent a disaster to a patient. I have found the LMA one of few innovations that have revolutionised my practice and would hate to see failure to carry out a 'preflight' checking procedure used to condemn it.

Freeman Hospital,
Newcastle upon Tyne NE7 7DN

I.D. CONACHER

'Bow-ties' in ITU

A 54-year-old man was admitted to our Intensive Care Unit after renal and respiratory complications following an aortic aneurysm repair. He required a 6-week period of ventilation during which a tracheostomy was carried out; he was then weaned slowly from the ventilator. The patient had a rather short neck which made attaching a T-piece to his tracheostomy tube difficult and once attached the T-piece was very liable to become separated. It was found that two elastic bands inserted through the flanges of the tracheostomy tube and onto the ends of the T-piece provided good support and prevented inadvertent disconnection. (Fig. 1). This rather elaborate 'bow-tie' arrangement saw our patient through another stage in his eventual recovery.

Glasgow Royal Infirmary,
Castle Street,
Glasgow

F.M.M. BRYDEN
F.W. DAVIES

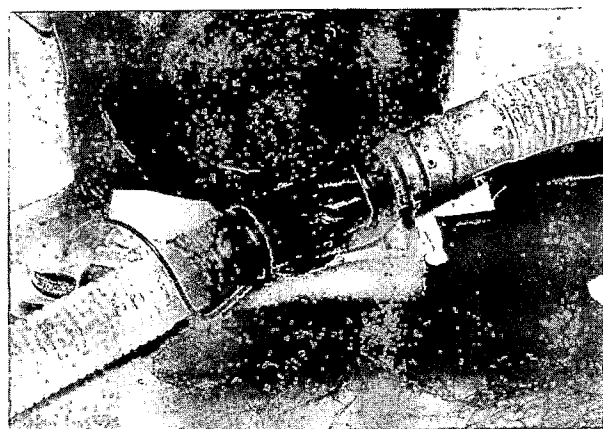


Fig. 1. Elastic bands attaching tracheostomy tube to T-piece.

Hazard Notice

Sengstaken (oesophageal varices) tubes manufactured by Warne Surgical Products HC (Hazard) 90:40. These tubes have been withdrawn following tests that have shown that the gastric balloons are prone to premature ageing, which could lead to failure of the product in use.

Safety Action Bulletin

Flanged curved arterial cannulae (all codes) manufactured by DLP incorporated (SAB (90) 72)

There is a potential risk of these arterial cannulae fracturing at the base of the curved tip. All cannulae manufactured before May 1989 have been withdrawn. Bio Medical Systems Ltd should be informed of any unused stock.

Standard and special order monitoring kit with Edwards disposable pressure transducer, manufactured by Baxter Edwards Critical Care (SAB (90) 73)

There has been a problem with transducer pressure drift manifested by either an increase or decrease in baseline thus leading to incorrect readings of pressure measurements. The manufacturers have decided to recall model numbers 51 and 53 manufactured from January 1990.

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THE ASSOCIATION OF ANAESTHETISTS OF GREAT BRITAIN AND IRELAND

APPLICATION FOR MEMBERSHIP

to

The Association of Anaesthetists of Great Britain and Ireland
9 Bedford Square,
London WC1B 3RA, England

(This application will be considered by Council at its next meeting. NO MONEY should be sent in the meantime. After acceptance of the application by Council, the Hon. Secretary will notify the applicant of Council's decision and the amount of subscription payable in the current financial year.)

.....19

To the Honorary Secretary,
Association of Anaesthetists of Great Britain and Ireland

I (full name) offer my name as a candidate

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for (a) ORDINARY MEMBERSHIP (b) OVERSEAS MEMBERSHIP (c) ASSOCIATE MEMBERSHIP (d) JUNIOR MEMBERSHIP of the Association (*please delete categories not applicable*)*

I hold the following appointment(s) as an anaesthetist (*please state grade if in the National Health Service, and indicate the approximate proportion of time spent in the practice of anaesthesia*):

Qualifications (with dates).....

(Signed).....

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Corporate author

The Committee on Enzymes of the Scandinavian Society for Clinical Chemistry and Clinical Physiology. Recommended method for the determination of gamma-glutamyltransferase in blood. *Scandinavian Journal of Clinical Laboratory Investigation* 1976; **36**: 119–25.

Anonymous. Epidemiology for primary health care. *International Journal of Epidemiology* 1976; **5**: 224–5.

BOOKS AND OTHER MONOGRAPHS

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OSLER AG. Complement: mechanisms and functions. New York: Prentice-Hall, 1976.

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American Medical Association Department of Drugs. *AMA drug evaluations*, 3rd edn. New York: Publishing Sciences Group, 1977.

Editor, compiler, chairman as author

RHODES AJ, VAN ROOVEN CE, comps. *Textbook of virology: for students and practitioners of medicine and other health sciences*, 5th edn. Baltimore: The Williams & Wilkins Co., 1968.

Chapter in book

WEINSTEIN L, SWARTZ MN. Pathogenic properties of invading micro-organisms. In: SODEMAN WA Jr, SODEMAN WA, eds. *Pathologic physiology: mechanisms of disease*. Philadelphia: W.B. Saunders, 1974: 457–72.

Agency publication

National Center for Health Statistics. Acute conditions: incidence and associated disability, United States, July 1968–June 1969. Rockville, MD: National Center for Health Statistics, 1972. (*Vital and health statistics, Series 10: Data from the National Health Survey*. No. 69) [DHEW publication No. (HSM) 72-1036].

OTHER ARTICLES

Newspaper article

SHAFFER RA. Advances in chemistry are starting to unlock mysteries of the brain: discoveries could help to cure alcoholism and insomnia, explain mental illness. How the messengers work. *Wall Street Journal* 1977 Aug 12: 1(col 1), 10(col 1).

Magazine article

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The closing section, The Recipient, discusses the effects that conduction blocks may have on the baby, and reviews the lay literature covering consumer's views of epidural practice.

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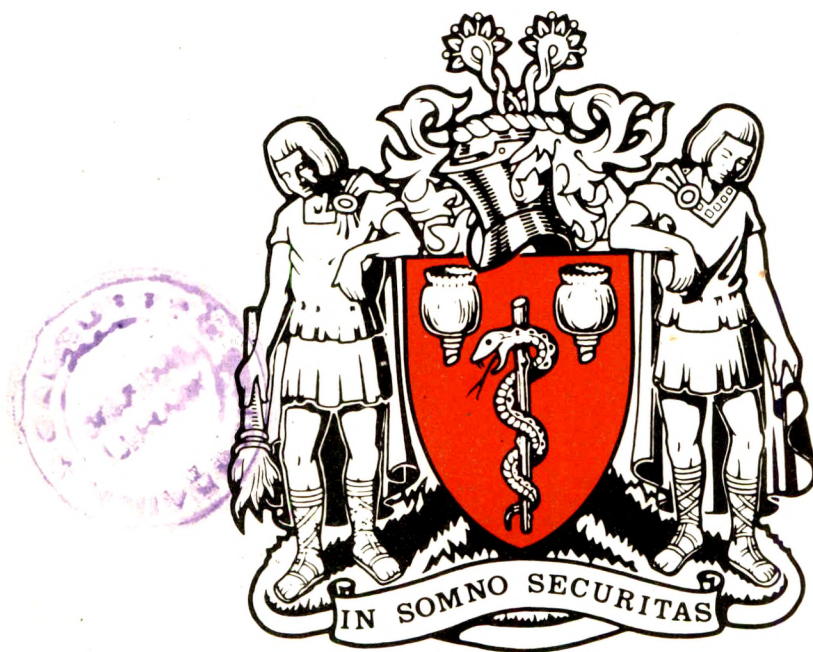


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Anaesthesia

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Volume 46 Number 3 March 1991



The Association of Anaesthetists of Great Britain and Ireland
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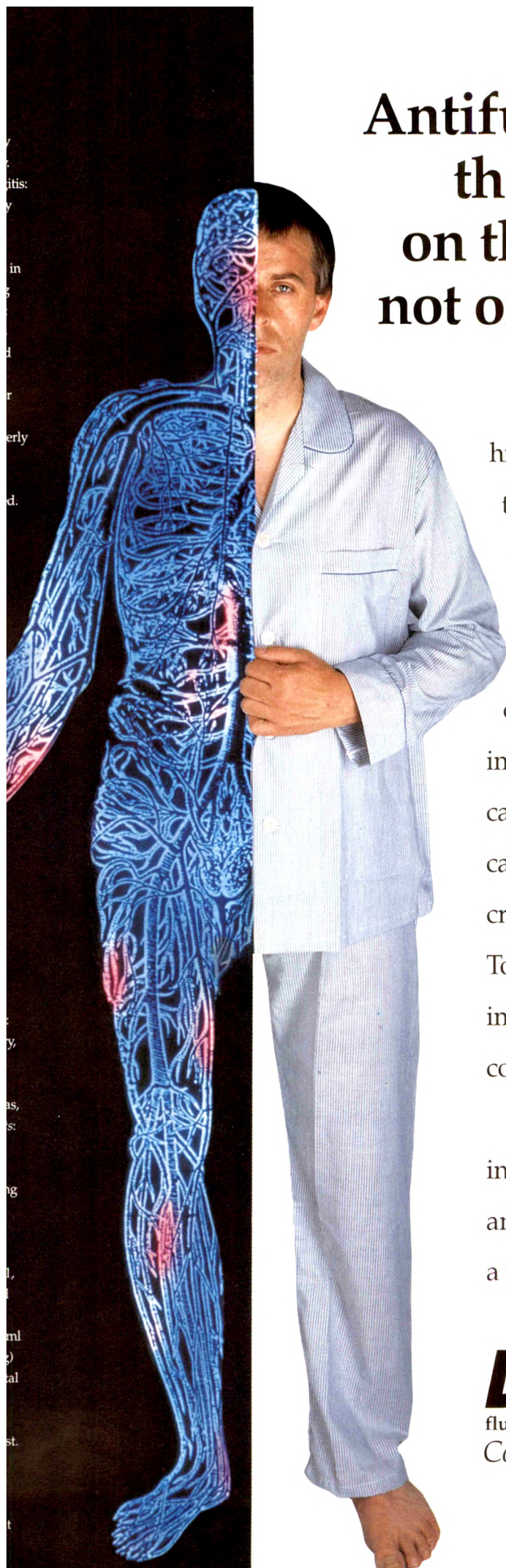
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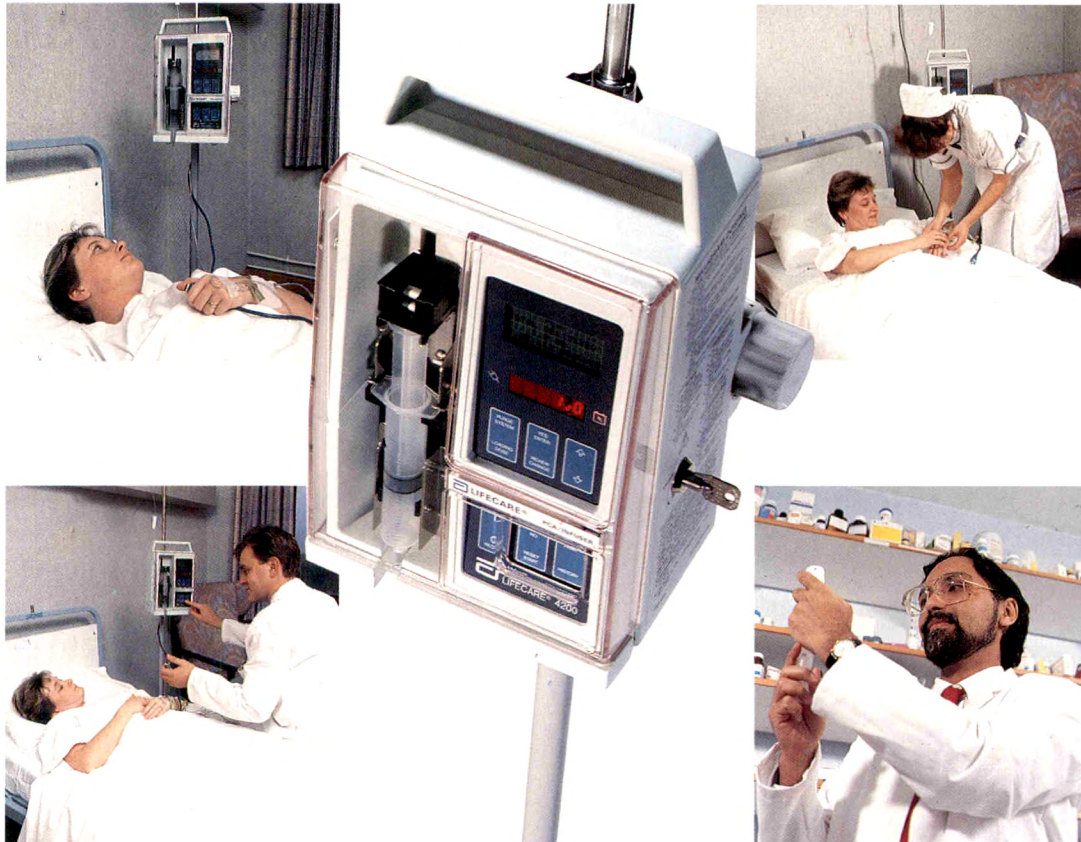
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Editorial

Postoperative pain relief—time to take our heads out of the sand?

If you think that sadomasochistic rituals are confined to video nasties viewed behind closed doors, then think again. A visit to most postoperative wards will show you the time-honoured ritual of inadequate postoperative pain management. Like most unpleasant things in life we have done our best to ignore the situation in the hope that it will disappear, but try as we may it will not pass us by and we must extract our heads from the sand and face the problem. Why should postoperative pain remain one of the unsolved problems of modern practice? Surely the major factor involved must be the attitude of staff and patients alike to postsurgical pain. In most aspects of medical care the patient enters hospital anticipating a successful outcome to their problem, and invariably such anticipation is well grounded. In the case of postoperative pain, patients expect ineffective pain relief and their carers ensure that they are not disappointed.¹

This has been the state of affairs in most people's living memory and sadly, save for the work of a few enthusiasts, the situation remains in the majority of hospitals in the United Kingdom. In most units the standard approach to postoperative analgesia is to delegate the task to the most junior member of the surgical staff who has little training, if any, in pain management and hence is often very wary of giving adequate quantities of drug. The inadequacy of dosing is further compounded by some nursing staff's reluctance to give analgesics to postoperative patients, and the problem of nurse availability particularly at night. From past studies²⁻⁴ it has been shown that as many as 75% of patients suffered moderate or severe pain whilst being treated with such a regimen.

It is against such a background that the Joint 'Colleges' Commission on the Provision of Surgical Services has published its report on pain after surgery.⁵ The document represents a very thorough exploration of the postoperative pain problem and gives guidance as to how the current situation might be improved without making its implementation impractical on financial grounds.

From today onwards every anaesthetist, surgeon and nurse should be encouraged to look at the standard of postoperative analgesic management currently offered to patients in their care and see how it can be improved. In most situations the initial improvement can be gained without expense by improving education of all involved with postoperative pain relief provision. The undergraduate teaching course in most medical schools is sadly lacking in instruction on pain relief. Hence the newly qualified doctor may have a wide knowledge of the many causes and treatments of subtle cardiac irregularities but has not the first idea of which drug should be used for postoperative pain or how much and how often it should be given. Such criticism levelled at undergraduate teaching must extend into postgraduate surgical training where the trainee surgeon is taught the skills of surgery but little, if anything, about the alleviation of postoperative pain. How often during

surgical ward rounds have you heard the surgeon enquire of the patient how their pain was? But let us not think that our haloes are set square above our heads. How often do we opt out and leave postoperative pain management to others? Do we ignore the patient once he (she) has left theatre and have no care for his (her) pain relief even in the recovery room? I fear that this is too often the case. Is our education on pain relief any better than our surgical colleagues? If one looks at the current standard anaesthetic textbooks used by trainees preparing for examinations, the content concerning acute pain management is often woefully small.

If doctors' prescribing habits could be improved, the system would still flounder if the prescribed drugs were not given by the nurses. Early teaching to nurses often overstates the dangers of analgesic drugs with regard to side effects and addiction potential, a situation resulting in reluctance to use analgesic drugs to their full advantage. Whilst accepting the dangers of opioid drugs, there can be no justification for leaving a patient suffering purely because the statutory 4 hours has not passed since the last dose.

Ongoing education of medical and nursing staff is essential to improve the care for the patient, but equally it is important to educate patients, after all they are suffering the pain. At present the patient expects pain and believes that little can be done about it, a situation that needs changing. If patients were made aware that pain could be adequately treated they might apply pressure for improvements to be made. With the introduction of 'open market' forces into health care will postoperative pain management become a feature of a hospital's attractiveness to patients and their general practitioners?

The Working Party Report⁵ suggests the establishment of an Acute Pain Service (APS) in all major hospitals. Such a service is based on the experience in the USA⁶ where a great deal of postoperative pain is dealt with by the Acute Pain Service which is funded on a fee payment basis. Such a fee payment system is not readily transferable to the National Health Service and some thought is needed as to how an APS might function in the UK. One group's experience in introducing an acute pain service is reported within this journal.⁷ They outline the problems that must be overcome in relation to improving postoperative pain management. In many respects they are fortunate in being a new hospital which has not yet become entrenched in time-honoured procedures, a situation that allows agreement between, and education of, all groups involved with postoperative management. It is clear from this example that the establishment of an APS within the hospital serves to highlight the problem, makes all staff more aware of any shortcomings and is supportive of any improvements.

In the short term it is likely that the APS will be set up in most hospitals by a few enthusiasts giving their own time to make the service viable, but in the long term adequate funding must be provided for the staffing

and support of such a service. Obviously the newly established APS cannot initially undertake to provide pain management of all postoperative patients but should concentrate on improving education of staff and patients, monitoring pain relief provision, establishing protocols, encouraging research and advising on problem patients. Whilst this is not the Rolls Royce service seen in the USA, large cars need fuel to make them run, sadly that is in short supply in the NHS.

Implementing all the aforementioned ideas would not have large financial implications, but the introduction of newer techniques of analgesic provision such as epidural opioids and patient-controlled analgesia (PCA) may have. Once again a comparison with the USA is inappropriate, since the cost of any technique there is passed to the patient (or their insurance company). However, research has shown that these newer techniques, by providing better levels of analgesia, can lead to better postoperative lung function,⁸ less pulmonary complications⁹ and, on average, to a shorter stay in hospital.¹⁰ Hence such systems may, whilst having an initial capital cost, eventually save money in shorter hospital stays.

The efficacy and cost of newer techniques is not the only issue surrounding their use, since the safety of these techniques has still not been fully clarified. Views vary on whether these techniques are safe on the general ward or whether a high dependency unit or intensive care unit are the correct places for their use. From the experience in the USA it seems that PCA is safe for general use, but the situation concerning epidural/spinal opioids is not so clear. Perhaps the best solution would be to change the outdated procedure of most hospitals whereby a patient returns from theatre to their pre-operative ward. Perhaps extended patient care should be implemented in a postoperative ward where all patients requiring analgesia would be admitted; in effect, an extended high dependency unit. This is an organisational problem which should not be insurmountable.

Now is the time to come out of hiding, shake the sand from our hair and tackle the problem for, as highlighted by Liebeskind and Melzack,¹¹ 'by any reasonable code, freedom from pain should be a basic human right,

limited only by our knowledge to achieve it'. We have the knowledge, sadly what we often lack is the will and finance to use it.

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Epidural infusion of diamorphine with bupivacaine in labour

A comparison with fentanyl and bupivacaine

G. R. ENEVER, H. A. NOBLE, D. KOLDITZ, S. VALENTINE AND T. A. THOMAS

Summary

We have compared the analgesic effects of three epidural infusions in a randomised, double-blind study of 61 mothers in labour. An initial dose of bupivacaine 0.5% 8 ml was followed by either bupivacaine 0.125%, bupivacaine 0.125% with diamorphine 0.0025% or bupivacaine 0.125% with fentanyl 0.0002%. All infusions were run at a rate of 7.5 ml/hour. Analgesia was significantly better in both the groups receiving opioids. Diamorphine was shown to be the more effective supplement to bupivacaine. The 5% incidence of pruritis in the opioid groups was less than that reported by earlier authors.

Key words

*Anaesthetic techniques, regional; epidural.
Analgesics; diamorphine, fentanyl.*

The epidural administration of opioids to women in labour has been widely investigated in recent years,¹ either as an alternative or as a supplement to local anaesthetic agents. Morphine has been found to be unsuitable² and pethidine requires large doses to be effective.³ Fentanyl has become popular,⁴⁻⁸ although early work indicated that analgesic quality might not be sustained into late labour.⁴ Alfentanil, sufentanil and butorphanol are currently under investigation¹ in the continued search for the ideal agent.

Unfortunately, epidural opioids have side effects.⁹ Nausea, pruritis and sedation are common, with respiratory depression a rare but serious complication. Problems are more likely if large doses are given, as when opioids are used alone. In order to reduce opioid dosage, combination with a local anaesthetic agent, such as bupivacaine, is employed. The quality of analgesia is generally thought to be better with these mixtures than with either drug alone. However, side effects, especially pruritis, are still noted in many reports. The most suitable combination and dose of opioid and local anaesthetic is yet to be identified.

Diamorphine has been popular for many years for post-operative analgesia via the epidural route.¹⁰ It is highly lipophilic, a property shared with fentanyl, but it has a longer duration of action. The only study of its use during labour to date reports the effects of a 5 mg bolus of diamorphine, where good analgesia was noted but also a 70% incidence of pruritis.¹¹ The paucity of published research is perhaps because most of the studies using epidural opioids in labour have come from countries where

diamorphine is not freely available, especially the United States.

In this study it was our intention to investigate the efficacy of diamorphine, combined with bupivacaine infused epidurally during labour. Groups receiving plain bupivacaine or a combination with fentanyl were included for comparison. We used smaller doses of both local anaesthetic and opioid agents than previously reported, to observe the effects on analgesia and the incidence of side effects. Infusions rather than an intermittent technique were used because we believe they are safer and more effective.^{12,13}

Methods

The study was approved by the District Ethics Committee, and written informed consent was obtained from all participants. Healthy mothers with uncomplicated singleton pregnancies were included in the study, provided they had not received any systemic opioid before requesting an epidural for analgesia. Women with a cervical dilatation of 6 cm or more at their previous vaginal examination, less than 150 cm tall or who weighed more than 100 kg at their last visit to clinic were not studied.

A large-bore intravenous cannula was inserted before performing the epidural and the circulation preloaded with 1000 ml compound sodium lactate solution. A baseline pain score was obtained at the peak of a contraction using a 20-cm visual analogue scale, a method validated¹⁴ and

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used in previous studies.^{15,16} The epidural catheter was inserted at the second or third lumbar interspace with the mother in the lateral position. Loss of resistance was detected with air and approximately 3 cm of catheter was inserted into the epidural space. All patients then received a test dose of 3 ml 0.5% bupivacaine followed by 5 ml to establish analgesia, with further increments of 3 ml given if necessary. If adequate analgesia was not obtained, the catheter was resited and the patient removed from the study.

A pain score and the extent of sensory and motor blockade were assessed and recorded before starting the infusions. Loss of temperature sensation to ethyl chloride spray was used to define the upper limit of sensory blockade, and motor blockade was assessed using the Bromage scale.¹⁷

Epidural infusions were commenced once adequate pain relief was established, with one of three randomly allocated solutions. Group P received plain 0.125% bupivacaine, Group D 0.125% bupivacaine with 0.0025% diamorphine and Group F 0.125% bupivacaine with 0.0002% fentanyl. All infusions were run at a rate of 7.5 ml/hour. They were prepared by the anaesthetist performing the epidural using 30 ml 0.25% bupivacaine diluted in a syringe to a final volume of 60 ml using 0.9% saline, after the addition of diamorphine 1.5 mg or fentanyl 120 µg. The infusions were given via a bacterial filter (Portex) using a syringe driver (Vickers Treonic IP4) and were continued until delivery was completed.

After starting the study infusions, specially trained midwives¹⁸ carried out all observations, unaware of which solution the mother received. Assessment of motor blockade, height of blockade and pain scores were repeated every hour. Arterial blood pressure, pulse rate, respiratory rate and fetal heart rate were recorded every 30 minutes. Posture of the mothers during the study was not controlled, as long as the position adopted avoided aortocaval compression. Respiratory rate was monitored for a further 8 hours after delivery.

If any mother complained of inadequate analgesia, additional top-up doses of 0.25% bupivacaine 8 ml were given. The number of doses was recorded, as was the time to first top-up. The top-up interval (being the infusion time divided by the sum of the top-up doses plus the initial dose)¹⁹ was obtained for each patient and the mean calculated for each group.

The incidence of pruritis, nausea or vomiting was elicited by direct questioning during and after the period of infusion.

Details of the mode of delivery and the condition of the infant, as assessed by the Apgar score, were recorded. The mothers were interviewed the next day to assess any urinary problems, and the general quality and acceptability of the epidural analgesia they had received.

Statistical analyses were performed using the Chi-squared test and analysis of variance (ANOVA) as appropriate.

Results

Sixty-one women were included in this study, of whom 21 received bupivacaine alone (group P), 19 bupivacaine with diamorphine (group D) and 21 bupivacaine with fentanyl (group F). There were no statistical differences in the demographic data of the three groups (Table 1).

Seven mothers in each group required no additional top-ups, while only four patients in the study needed more than three (Table 2). The times (SD) to either first top-up or delivery in the three groups were 211 (106) minutes (group P), 252 (139) minutes (group D) and 260 (152) minutes (group F). There was no statistical difference between the three groups.

There was no statistical difference in the mean top-up interval between the three groups, the values being 192 (SD103) minutes (group P), 213 (SD144) minutes (group D) and 233 (SD142) minutes (group F).

The mean duration (SD) of infusion for the three groups was 327 (148) minutes (group P), 393 (204) minutes (group

Table 1. Demographic data on entering study; mean (SD). There were no significant differences between the three groups ($p < 0.05$).

	Bupivacaine (<i>n</i> = 21)	Bupivacaine/ diamorphine (<i>n</i> = 19)	Bupivacaine/ fentanyl (<i>n</i> = 21)
Age; years	27.1 (6.8)	24.3 (6.5)	23.7 (3.9)
Height; cm	161(7)	160(6)	162(5)
Weight; kg	74.0(12.0)	67.0(10.3)	74.4(13.8)
Primiparous; %	66.7	68.4	80.9
Cervical dilatation; cm	3.2 (1.3)	3.6 (1.6)	3.2 (1.4)
Median pain score (Range)	153 (65–200)	158 (105–200)	145 (88–20)

Table 2. Requirements for top-up injections of bupivacaine during infusion; number of patients (%). There were no significant differences between the three groups ($p < 0.05$).

	Bupivacaine (<i>n</i> = 21)	Bupivacaine/ diamorphine (<i>n</i> = 19)	Bupivacaine/ fentanyl (<i>n</i> = 21)
No top-ups	7(33.2)	7(36.8)	7(33.2)
One top-up	9(43.0)	4(21.1)	5(23.8)
Two top-ups	1 (4.8)	5(26.3)	5(23.8)
Three top-ups	4(19.2)	1 (5.3)	2 (9.5)
More than three top-ups	0	2(10.5)	2 (9.5)

D) and 468 (195) minutes (group F); the difference between groups P and F achieved statistical significance ($p < 0.02$). However, if only the primiparous mothers were compared, then the mean duration of infusion for the groups was 369 minutes (P), 457 minutes (D) and 457 minutes (F); there was no statistical difference between them.

The hourly median visual-analogue pain scores (0–200) for each group are presented with the range of scores and number of patients remaining in the group (Table 3). The median pain scores in group D were consistently low, and the difference when compared to the other two groups was statistically significant ($p < 0.05$).

The median pain scores before delivery for all the women studied are presented (Table 3), with the scores for those with longer infusion times (over 6 hours) recorded separately in order to assess the effect of duration on the quality of analgesia. With the longer infusion times, the group F patients had significantly worse pain scores ($p < 0.01$) than the other two groups.

Hourly pain scores were also grouped under the following headings; no pain, score of 0; mild pain, score of 1 to 60 and moderate/severe pain, score of 61 to 200. The scores were then summed for each group, and presented as a bar chart (Fig. 1). Both groups D and F had more pain-free hours than group P (group D, $p < 0.001$ and group F, $p < 0.02$).

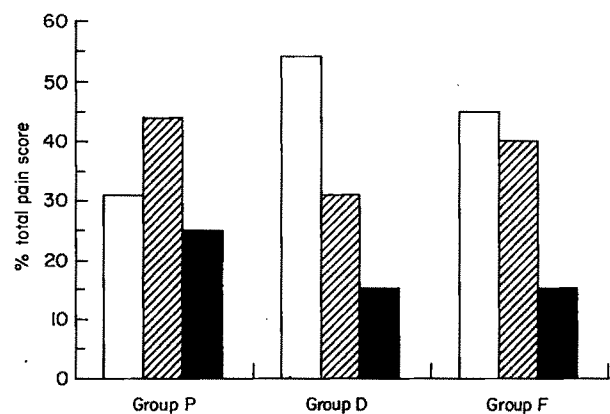


Fig. 1. Distribution of total hourly pain scores for the three groups. Both group D bupivacaine/diamorphine ($p < 0.001$) and Group F bupivacaine/fentanyl ($p < 0.01$) have more scores = 0 than Group P, bupivacaine above. □, scores of zero; ▨, scores of 1–60; ■, scores of 61–200.

The median level of sensory blockade in all groups was T₉, and the highest level in any woman was T₅. Motor blockade was similar in all three groups (Table 4). Urinary problems during labour were difficult to assess, because it was common practice to catheterise patients before operative delivery. However, the incidence of problems after

Table 3. Median pain scores (range 0–200) for the three groups during labour. Scores at delivery for all patients, and for those with infusion times over 6 hours are also given below; n = number remaining undelivered in each group. Scores for group D were significantly lower ($p < 0.05$).

Median pain scores during labour						
Infusion time (hours)	Bupivacaine		Bupivacaine/ diamorphine		Bupivacaine/ fentanyl	
		n		n		n
1	5 (0–118)	21	0 (0–190)	19	3 (0–86)	21
2	14 (0–146)	21	0 (0–140)	18	0 (0–85)	21
3	12 (0–140)	19	1 (0–122)	16	0 (0–129)	20
4	25 (0–125)	13	2.5 (0–119)	14	0 (0–66)	18
5	27 (0–160)	12	4.5 (0–179)	11	30 (0–115)	13
6	80 (0–170)	7	5 (0–195)	9	5 (0–60)	11
7	18.5 (6–50)	4	0 (0–30)	5	12 (0–71)	6
8	5 (2–8)	2	0	1	60 (8–135)	5
9	75 (0–150)	2	7.5	1	60 (24–106)	3
10	75 (0–150)	2	0	1	95 (60–165)	3
11	0	1	—	0	55 (30–80)	2
12	—	0	—	0	101 (65–138)	2
Median pain scores at delivery						
All patients	21 (0–170)	21	0 (0–195)	19	38 (0–165)	21
Patients with infusions > 6 hours	3 (0–12)	4	0 (0–30)	5	103* (16–165)	6

* $p < 0.01$

Table 4. Incidence of motor blockade, using Bromage scale (4 = no blockade, 1 = full blockade); number of patients (%). There were no significant differences between the three groups.

Mean motor blockade (both legs)	Bupivacaine ($n = 21$)	Bupivacaine/ diamorphine ($n = 19$)	Bupivacaine/ fentanyl ($n = 21$)
Grade 4	4 (19.2)	6 (31.6)	5 (23.8)
Grade 3 or above	13 (61.6)	7 (36.8)	11 (52.4)
Less than grade 3 (either leg)	4 (19.2)	6 (31.6)	5 (23.8)

Table 5. Incidence of side effects; number of patients (%) and median levels of sensory blockade. Mode of delivery; number of patients (%). There were no significant differences between the three groups.

	Bupivacaine (n = 21)	Bupivacaine/ diamorphine (n = 19)	Bupivacaine/ fentanyl (n = 21)
Decrease in systolic blood pressure (>20%)	4(19.2)	5(26.3)	5(23.8)
Nausea only	2 (9.5)	0	0
Nausea with vomiting	2 (9.5)	4(21.1)	3(14.3)
Pruritis	0	1 (5.3)	1 (4.6)
Apgar <8 at 1 minute	5(23.8)	4(21.1)	4(19.2)
Apgar <8 at 5 minutes	2 (9.5)	1 (5.3)	3(14.3)
Sensory level	T ₉	T ₉	T ₉
Spontaneous vaginal	6(28.6)	10(52.6)	6(28.6)
Forceps	6(28.6)	5(26.3)	5(23.8)
Ventouse	6(28.6)	4(21.1)	7(33.3)
Caesarean section	3(14.2)	0	3(14.3)

delivery was very low, with only one patient in each group requiring short periods of catheterisation.

No symptoms associated with systemic toxicity of bupivacaine were noted during this study. In group D the dose rate of diamorphine was 0.163 mg/hour and the mean total dose per patient was 1.05 mg. In group F the dose rate of fentanyl was 15 µg/hour and the mean total dose per patient was 117 µg. No evidence of respiratory depression, as judged by respiratory rate changes, was noted in any mother. No sedation was noted in any patient, apart from that normally seen when pain is relieved during labour.

The incidence of other side effects are shown in Table 5. There was only one woman in each of group D and group F who experienced pruritis. There was no significant difference in the condition of infants at one or five minutes, and no infants required naloxone or resuscitation other than normal bag and mask oxygen.

The mode of delivery is also shown in Table 5. There was no statistically significant difference between the three groups, although there were more spontaneous vaginal deliveries in group D ($0.1 > p > 0.05$).

Discussion

In this study, we have demonstrated that the addition of only small amounts of opioids to epidural solutions improved the quality of analgesia. This was not unexpected with the fentanyl group because we had the results of previous studies for comparison.^{7,8} However, it was interesting to find that analgesia in the diamorphine group was as good as, or better than, the fentanyl group. In fact, the women receiving diamorphine had more hourly pain scores of zero than the other two groups, and this was achieved with a dose of only 0.163 mg/hour. The need for top-ups was not increased by our low infusion rates when compared to other fixed-rate studies.^{12,20} Our mean top-up interval is similar to that found by other investigators who used larger doses of bupivacaine.¹⁹ The interval exceeded 3 hours for all three groups.

For an infusion regimen to offer benefits to the mother in labour, we believe that it is important for it to be effective into the second stage and during delivery. For this reason we continued our infusion until the third stage of labour was completed and continued our observations until delivery was imminent. By doing so we noted a tendency for pain scores to increase in patients receiving fentanyl when labour lasted over 6 hours. Although only 15 patients out

of the 61 had long labours (Table 3), this difference achieved statistical significance at $p = 0.01$. This finding was in keeping with some previous research,⁴ where inadequate analgesia was seen with repeated bolus doses of fentanyl. Whether the pattern we saw was a result of our dose of fentanyl being inadequate to compensate for the increasing pain of late labour, or that we were observing tachyphylaxis we are not sure. The fact that the group receiving no opioids at all had lower pain scores than the fentanyl group in late labour leads us to surmise that the latter may be true. The diamorphine group, however, maintained a consistent quality of analgesia in the majority of patients receiving it. This phenomenon deserves further investigation.

There was a low incidence of pruritis in this study, with only one woman in each of the diamorphine and fentanyl groups reporting it. This is an improvement on previous investigations using either epidural fentanyl,^{4,5,20-23} where the incidence ranged between 17 and 75%, or epidural diamorphine, where it was 72% after a single bolus dose.¹¹ Our low incidence may well be related to the small doses of opioids given in this study. We avoided the use of opioids in the initial bolus dose, and used very low concentrations in our infusions. This may indicate that pruritis after epidural opioids is dose related.

The incidence of nausea and vomiting was similar to previous investigations, occurring in approximately 20% of women irrespective of their group. It is interesting to note that the incidence of nausea in women without epidurals is appreciable during labour,²⁴ often related to delayed gastric emptying and the use of oxytocic drugs.

No woman in this study developed a respiratory rate of less than 10 breaths/minute, as would be expected from previous studies.^{10,11,25} However, a recent report using long-term oximetry to monitor nonobstetric postoperative patients has indicated that respiratory depression can occur with epidural diamorphine,²⁶ although much larger doses were used than in our study. Oximetry, and perhaps even capnography, may play a role in future investigations of epidural opioids in the perinatal period, since the limitations of observing only respiratory rates are well known.

We saw no drowsiness in the women receiving diamorphine in labour compared to the other reported use of it in a similar situation.¹¹ We presume that, as we have seen analgesic effects but little or no evidence of side effects, that the opioids we are giving are acting only locally. This is in keeping with the marked lipophilicity combined with low

doses of both fentanyl and diamorphine. However, we cannot rule out the possibility that the opioids given were having some systemic action.

The groups receiving opioids in their epidurals in this study had longer infusion times. The fentanyl and diamorphine groups have identical mean times, when just the primiparous patients are compared, 88 minutes longer than the plain group. Variability causes this difference to not achieve statistical significance, but it is an interesting trend. It differs from a previous report⁷ in which women receiving epidural fentanyl had shorter labours than those who received plain local anaesthetic. It is possible that epidural opioids may be acting to alter the neurohumoral control of uterine activity. Evidence to support this theory is meagre, although plasma oxytocin levels at delivery have been shown to be reduced by local anaesthetics,²⁷ presumably by neural blockade, while recent work has indicated that epidural fentanyl may affect uterine activity.²⁸

This study has indicated that it is possible to obtain adequate epidural analgesia by using low dose solutions of combined local anaesthetic and opioids infused at a low rate. We believe this combination improves safety and reduces the incidence of some side effects. We have also investigated the use of low doses of diamorphine during labour and have found it to be effective as a supplement to bupivacaine.

Acknowledgments

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The alkalinisation of bupivacaine for intercostal nerve blockade

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Summary

A double-blind randomised study was performed to investigate the effect of pH adjustment of bupivacaine, with adrenaline 1 : 200 000, on the duration of block and pain relief after intercostal nerve blockade following thoracotomy. One group (n = 10) received bupivacaine with adrenaline 1 : 200 000 (pH = 4.1) and the other (n = 10) received alkalinised bupivacaine with adrenaline 1 : 200 000 (pH = 6.9). There was no significant difference in block duration (mean 23.9 and 26.4 hours respectively) visual analogue pain scores or morphine usage. Patients were more likely to have a block during the first 12 hours if they received alkalinised bupivacaine ($p < 0.01$, Chi-squared test). A progressive regression of block, not previously described, was observed, explicable by means of spread of local anaesthesia to adjacent intercostal nerves. Alkalinisation of bupivacaine with adrenaline for intercostal nerve blockade has little clinical benefit.

Key words

*Anaesthetic techniques, regional; intercostal nerve block.
Anaesthetics, local; bupivacaine, alkalinisation.*

Intercostal nerve blockade with local anaesthetic is frequently used to provide analgesia after thoracotomy.^{1–4} Its principal advantages are those of simplicity and effectiveness with few complications.³ However, the relatively short duration of action of local anaesthetics limits the usefulness of a single injection technique. Various methods have been employed to extend the duration of action; adrenaline has been some success⁵ as has the use of an intercostal catheter.⁶

Recently, addition of sodium bicarbonate to local anaesthetics has been shown to increase their duration of action at various sites; epidural,⁷ sciatic,⁸ brachial plexus,⁹ and intravenous.¹⁰ Other studies have failed to demonstrate this effect.¹¹ The prolongation is said to occur because of a mass effect; more drug is in its uncharged lipid soluble form as a result of the increase in pH and therefore more reaches the site of action inside the nerve membrane.^{12,13}

The purpose of this randomised, double-blind study was to assess the effects on block duration and quality of analgesia of raising the pH of bupivacaine with adrenaline by the addition of sodium bicarbonate for intercostal nerve blockade after thoracotomy.

Methods

Approval for the study was obtained from the local ethics committee. Twenty patients undergoing thoracotomy were studied, having given full informed consent.

All patients received a standardised general anaesthetic. Premedication was with morphine 10 mg intramuscularly one hour before operation. Anaesthesia was induced with propofol and the trachea intubated following atracurium 0.6 mg/kg. Anaesthesia was maintained with enflurane (0.8–1.5%) in 50% nitrous oxide and 50% oxygen and neuromuscular blockade with an infusion of atracurium at a rate of 0.5 mg/kg/hour; the lungs were mechanically ventilated. The thoracotomy incision was made between the fourth and fifth ribs for pulmonary surgery and that for oesophageal surgery between the sixth and seventh ribs with an extension to the anterior abdomen.

Intercostal blocks from T₂ to T_{11–12} were performed at the angle of the ribs or medial to the border of the scapula immediately after induction of anaesthesia. The patients were randomly allocated to receive, in a double-blind fashion, either the usual or pH-adjusted bupivacaine, both

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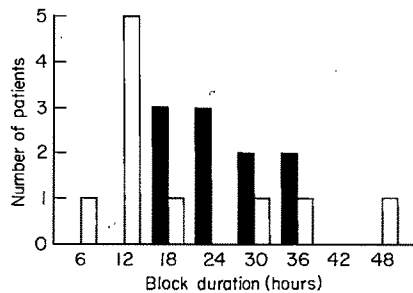


Fig. 1. Duration of block (hours) for bupivacaine and saline □, bupivacaine and alkali ■.

with adrenaline 1:200 000. Either 2.25 ml of normal saline (pH 4.1) or 2.25 ml 8.4% sodium bicarbonate (pH 6.9) were added to 10 ml of the bupivacaine solution; the percentage of uncharged drug is 0.1% and 7.1% respectively. No precipitation was seen in either solution after it had stood for 48 hours. Three ml of either of these solutions was injected in each space.

All the patients received routinely diclofenac 75 mg intramuscularly at induction of anaesthesia and twice daily after operation. A continuous infusion of morphine 1 mg/hour was also given and supplementary intramuscular morphine, 5 mg 2 hourly, was available as required.

The patients were assessed upon awakening and at 6, 12, 24 and 36 hours after induction. Analgesia was assessed subjectively after a deep inspiration using a 10-cm visual analogue score (VAS) with no pain at the left-hand side and the severest pain imaginable at the right. The extent of anaesthesia was assessed by pinprick using a short-bevelled 27-gauge needle in the midclavicular line. The duration of block was defined as the time when analgesia was present only in the dermatomes overlying the thoracotomy incisions. Supplementary morphine usage for the first 48 hours was determined.

Statistical analysis was carried out using Student's unpaired *t*-test for the patient demographic data and the Mann-Whitney *U* test for analysis of VAS, block duration and morphine usage. The Chi-squared test was used to examine the difference in the number of subjects with a block at the assessment times.

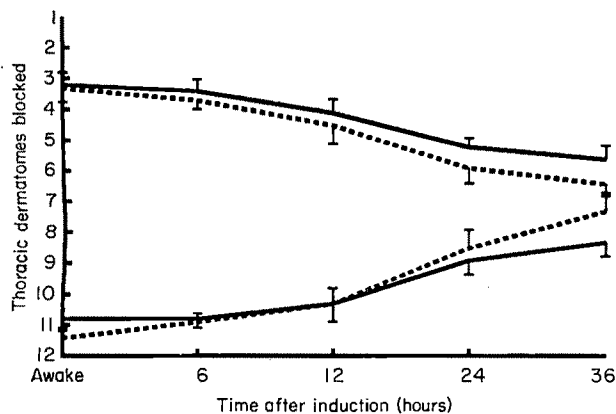


Fig. 2. Extent (mean, SD) of dermatomal block, at assessment times (hours) for bupivacaine and saline ----, bupivacaine and alkali —.

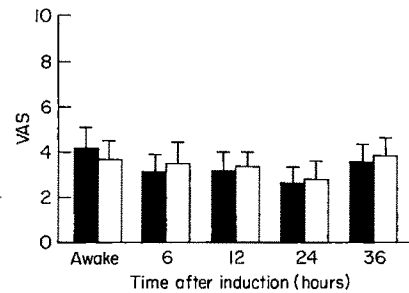


Fig. 3. Visual analogue scores (mean, SD) for bupivacaine and saline □, bupivacaine and alkali ■.

Results

Both groups were similar in respect to age, weight and height. There was a preponderance of males in the saline group. Both groups had similar operations (Table 1).

The mean block duration in the alkalinised group was 26.4 (SD 6.96) hours compared to 23.4 (SD 13.68) hours in the saline group (Fig. 1). A block was more likely to be present during the first 12 hours in those who had received alkalinised bupivacaine ($p < 0.01$).

The extent of the block regressed gradually from the upper and lower segments towards the central segments, T₇ and T₈. Recovery was incomplete in some patients even after 30 hours. Regression of the blocks tended to be slower in the alkalinised group although this was not statistically significant (Fig. 2).

The initial VAS scores were low on awakening and remained at a similar level throughout the assessments. No difference was seen between the two groups (Fig. 3). There was no significant difference between the two groups in the amount of supplementary morphine required by patients.

Discussion

This study demonstrates that the addition of sodium bicarbonate to 0.5% bupivacaine with 1 in 200 000 adrenaline for intercostal nerve blockade does not prolong duration of anaesthesia to pinprick. This is in contrast to the other studies which have looked at the effects in the epidural space, the sciatic nerve, the brachial plexus and intravenous regional anaesthesia. One reason for the poor effect seen here may be because of the greater systemic absorption of bupivacaine from the intercostal space compared to the

Table 1. Patient demographic data; mean (SD) and operations performed.

	Bupivacaine with saline (n = 10)	Bupivacaine with alkali (n = 10)
Male	7	5
Female	3	5
Age; years	53.7 (22.5)	53.5 (20.7)
Weight; kg	65.7 (12.3)	68.7 (13.9)
Height; m	1.72 (0.08)	1.71 (0.08)
Pleurectomy	2	3
Thoracotomy	6	6
Oesophageal surgery	2	1

other sites.^{14,15} This greater absorption may prevent the mass effect from occurring.

To our knowledge only two other studies have assessed the duration and spread of intercostal nerve blockade using analgesia to pinprick.^{1,2} Most studies have relied on patients' reports of pain or analgesic requirement.^{4,16} We have shown that the duration of anaesthesia to pinprick is much longer than previously reported, with a combined mean duration of 24.9 hours compared with 11 and 16.3 hours. Three reasons may explain this discrepancy: firstly, the patients in this study had a greater total mass of drug given (30 ml of 0.5% bupivacaine compared with 16 and 20 ml); secondly, a morphine infusion was given. Systemic morphine can increase the dermatomal extent of sensory analgesia to pinprick after regression of block during epidural bupivacaine infusion. This has been attributed to stimulation, by morphine, of brain stem neurones that have descending pathways to the spinal level and which may inhibit pain transmission.¹⁷ Equally, systemic morphine might stimulate opiate receptors directly in the dorsal horn and block onward cephalad transmission of action potentials in pain pathways; thirdly, the blocks were performed before surgery in contrast to the other studies, and after an opioid premedication. It is possible that analgesia may be enhanced by preventing central nervous memory of pain^{18,19} or by suppressing central sensitisation of dorsal horn neurones.²⁰

The gradual regression of the blocks from the extremes to the middle was an unexpected observation and has not been described before. The long duration of the T₅ and T₆ blocks has been ascribed to nerve damage during suturing of the chest wall and impairment of vascular absorption of the local anaesthetic.^{1,2} However, the progressive nature of the regression makes it more likely that extrapleural or paravertebral spread is occurring.^{16,21-23} Recent studies have failed to demonstrate consistent spread of small volumes of solution from the intercostal space injected. Patients given 3 ml of a bupivacaine-methylene blue mixture²⁴ or volunteers injected with 5 ml lignocaine-radiopaque dye mixture²⁵ showed no spread. However, the spread was assessed soon after injection, at 13 and 2 minutes respectively. Spread may be slow and only seen after a much longer time. It is also recognised that there is overlapping of dermatomal nerve supply,²⁶ so it may be advisable to perform multiple blocks above and below the nerves that are required to be blocked to obtain both full anaesthesia and a longer duration of action.

In conclusion, alkalisation of bupivacaine with adrenaline in intercostal nerve blockade increases the likelihood of block during the first 12 hours, but does not significantly prolong duration of block and does not appear to confer clinical benefit if used as part of a balanced analgesic regimen.

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Intravenous lignocaine and sympathoadrenal responses to laryngoscopy and intubation

The effect of varying time of injection

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Summary

We have studied the effect of varying the timing of a prior dose of intravenous lignocaine 1.5 mg/kg on the cardiovascular and catecholamine responses to tracheal intubation. Forty healthy patients were given an intravenous injection of either placebo or lignocaine 2, 3 or 4 minutes before tracheal intubation. There was a significant increase in heart rate of 21–26% in all groups. There was no significant increase in mean arterial pressure in response to intubation in any group of patients given lignocaine before intubation, but in the placebo group, mean arterial pressure increased by 19.1% compared to baseline values ($p < 0.05$).

Key words

*Intubation, tracheal; complications.
Anaesthetics, local; lignocaine.*

The circulatory response to laryngoscopy and intubation was described first by King in 1951.¹ This response may be harmful in some patients, and many techniques have been devised to reduce the extent of the response, including the use of adrenergic blockers,² vasodilators,³ high dose opioids,⁴ deep inhalational anaesthesia,¹ and lignocaine, administered intravenously^{5–12} or topically.^{7,8,13}

Theoretically the use of lignocaine may be advantageous since it should avoid prolonged recovery and cardiovascular complications. However, recent studies have drawn conflicting conclusions on its efficacy.^{6,7,9} This may be explained partly by differences in the interval between administration of lignocaine and tracheal intubation.⁶ We have therefore examined this aspect in a double-blind study of healthy patients.

Methods

Approval was given by the District Ethics Committee. Informed consent was obtained from 40 ASA class 1 patients scheduled to undergo gynaecological surgery requiring tracheal intubation.

Premedication comprised morphine 0.15 mg/kg and prochlorperazine 0.2 mg/kg intramuscularly one hour before operation. On arrival in the anaesthetic room, electrocardiogram (ECG) monitoring and noninvasive arterial pressure recording (Datex Cardiocap) were instituted; an intravenous cannula was inserted into a vein on the dorsum of the hand and another was placed in the

contralateral antecubital fossa. A period of 5 minutes' stabilisation was allowed to elapse. Measurements of baseline heart rate and arterial pressure were recorded, and a sample of blood was taken into a lithium heparin tube for assay of plasma concentrations of catecholamines. Patients were allocated randomly to one of four groups. Those in group 1 received intravenous isotonic saline 2, 3 or 4 minutes before tracheal intubation. Patients in groups 2, 3 and 4 received intravenous lignocaine 1.5 mg/kg 2, 3 or 4 minutes respectively before intubation. All injections were given over 10 seconds.

Anaesthesia was induced with thiopentone 4–5 mg/kg (followed by vecuronium 0.1 mg/kg) and maintained with nitrous oxide 66% and enflurane 1% in oxygen. The patient's lungs were ventilated manually via a Bain system using a fresh gas flow of 90 ml/kg. End-tidal CO₂ concentration (Datex Cardiocap) was maintained at 4.5–5.0%. The trial drug was given at the appropriate time before intubation, which took place 4 minutes after induction in all patients.

Laryngoscopy (Macintosh blade) and intubation (8.5 mm i.d. Portex tube) were performed by one of two consultant anaesthetists, unaware of the group to which the patient belonged. Measurement of heart rate and arterial pressure and further blood samples were taken at 1, 3 and 5 minutes after tracheal intubation. Blood was centrifuged at 4°C and the separated plasma stored at –70°C until analysis for catecholamine concentrations by high pressure liquid chromatography as described elsewhere.¹⁴

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Table 1. Patient data, mean (SD).

	Group 1 n = 10	Group 2 n = 10	Group 3 n = 10	Group 4 n = 10
Age; years	30.6 (5.7)	34.1 (8.9)	30.9 (5.4)	32.5 (3.5)
Weight; kg	65.4 (6.3)	62.6 (8.2)	67.9 (10.1)	63.9 (6.3)
Height; cm	164.6 (5.4)	160.8 (3.8)	162.1 (3.5)	162.4 (3.5)

The data were analysed statistically by ANOVA followed by Duncan's multiple-range tests.

Results

The groups were comparable in respect of age, weight, height and baseline values of heart rate, arterial pressure and plasma catecholamine concentrations. (Tables 1 and 2).

Intubation of the trachea was always completed within 15 seconds (defined as the time from insertion to removal of the laryngoscope). The cardiovascular and catecholamine data for the four groups are shown in Table 2. Technical problems resulted in the loss of results for four catecholamine assays.

The increases in heart rate at one minute were significant ($p < 0.05$) in all groups, with no differences between the groups. Heart rate increases were no longer significant by 3 minutes; the only between-group difference was at 5

Table 2. Cardiovascular variables and plasma and catecholamine concentrations, mean (SD).

		Baseline	1 minute	3 minutes	5 minutes
Heart rate; beats/minute	Group 1	81.8 (18.3)	96.7* (11.7)	86.0 (10.4)	76.7 (7.3)
	Group 2	84.4 (18.3)	99.0* (12.6)	82.3 (9.2)	73.4 (7.0)
	Group 3	77.7 (12.9)	95.9* (13.9)	80.6 (7.6)	75.6 (8.5)
	Group 4	80.3 (12.0)	99.1* (12.6)	88.8 (8.9)	84.1† (8.5)
Mean arterial pressure; mmHg	Group 1	98.6 (13.9)	116.3* (13.9)	96.9 (9.2)	85.7 (7.6)
	Group 2	93.2 (9.8)	100.1 (13.3)	92.2 (7.9)	77.7 (4.4)
	Group 3	95.0 (12.3)	97.6 (15.8)	91.1 (7.9)	83.7 (9.8)
	Group 4	89.4 (8.9)	88.4 (14.2)	87.9 (14.2)	79.9 (12.0)
Adrenaline concentration; nmol/litre	Group 1	0.51 (0.15)	0.48 (0.18)	0.38 (0.15)	0.44 (0.21)
	Group 2	0.56 (0.32)	0.40 (0.16)	0.48 (0.47)	0.42 (0.25)
	Group 3	0.48 (0.21)	0.57 (0.33)	0.49 (0.39)	0.37 (0.18)
	Group 4	0.52 (0.24)	0.68 (0.39)	0.51 (0.21)	0.54 (0.30)
Noradrenaline concentration; nmol/litre	Group 1	2.87 (1.49)	3.62 (2.24)	3.05 (3.13)	3.29 (2.72)
	Group 2	2.41 (0.63)	3.02* (0.66)	2.66 (0.85)	2.57 (0.94)
	Group 3	2.72 (1.05)	3.51 (1.59)	3.15 (1.59)	2.72 (1.18)
	Group 4	3.40 (1.55)	4.15 (2.56)	3.37 (1.33)	3.31 (1.23)

*Significantly greater than baseline ($p < 0.05$).

†Significantly greater than other groups ($p < 0.05$).

Table 3. Summary of previous studies on intravenous lignocaine and responses to intubation.

Study	Number of patients	Patient characteristics	Dose of lignocaine mg/kg	Interval between lignocaine and intubation (minutes)	Attenuation of increases in	
					Arterial pressure	Heart rate
Hamill <i>et al.</i> ⁸	22	Intracranial tumours	1.5	1	No	No
Abou-Madi <i>et al.</i> ⁵	30	No exclusions	0.75	'Within 2-3 minutes'	Yes	Yes
		Age 28-85 including IHD+HT	1.5			
Stoelting ¹⁰	36	Cardiac patients excluded	1.5	1.5	Yes	No
Stoelting ⁷	24	CABG operations	1.5	1.5	No	No
Miller <i>et al.</i> ¹¹	45	Chinese, thoracic surgery	1.5	1, 2 and 3	No	No
Laurito <i>et al.</i> ¹	40	ASA 1 and 2	2	1.75	No	No
Tam <i>et al.</i> ⁶	30	Only excluded HT	1.5	1, 2, 5	No	No
				and 3	Yes	Yes
Chraemmer-Jorgensen <i>et al.</i> ⁹	16	No CVS or RS pathology	1.5	2	No	No
Denlinger <i>et al.</i> ¹²	20	Cardiac surgery	1.5	1.5	Yes	No
Present study	40	ASA 1	1.5	2, 3 and 4	Yes	No

IHD, ischaemic heart disease; HT, hypertension; CABG, coronary artery bypass graft; RS, respiratory system; CVS, cardiovascular system.

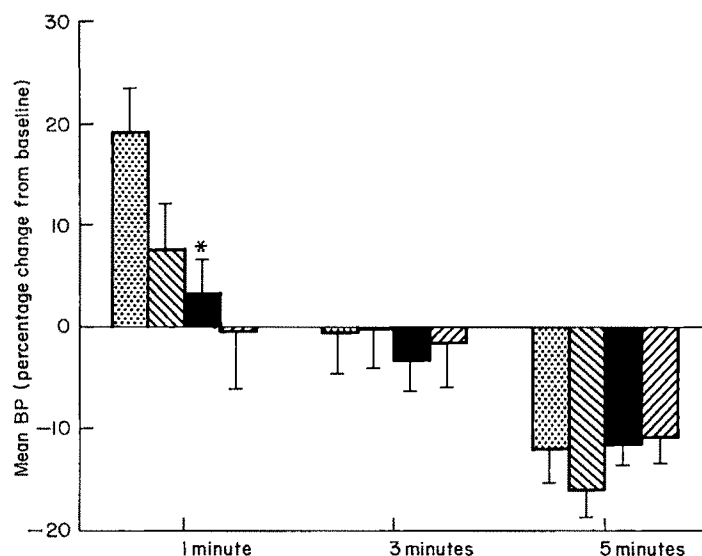


Fig. 1. Mean (SEM) percentage change in mean arterial pressure from baseline values at 1, 3 and 5 minutes after tracheal intubation. $n = 10$ in each group; □, group 1; ▨, group 2; ■, group 3; ▩, group 4. * $p < 0.05$ compared with placebo, group 1.

minutes when the heart rate in group 4 was significantly higher ($p < 0.05$) than that in the other three groups.

The only group in which mean arterial pressure (MAP) was significantly higher at one minute compared with awake values was group 1 ($p < 0.05$). The percentage increases in MAP at one minute in group 3 (+3.1%), and group 4 (-0.5%) were significantly different ($p < 0.05$) from that in group 1 (+19.1%). The only other significant difference between groups in respect of arterial pressure changes was between the diastolic pressures at one minute in group 1 (+27.86%) and group 4 (+3.9%) (Fig. 1).

There were no significant differences in noradrenaline or adrenaline concentrations between the groups at any time after intubation. There was an increase in noradrenaline concentration in all groups at one minute after intubation which failed to reach significance except in group 2 ($p < 0.05$).

Discussion

We have demonstrated that in this group of healthy patients, intravenous lignocaine 1.5 mg/kg given 4 minutes before laryngoscopy prevented an increase in mean arterial pressure, but when given 2 minutes before, lignocaine had no significant advantage over placebo. Heart rate increased to the same degree in all groups. Plasma catecholamine concentrations increased in all groups but the changes reached significance only in group 2 at one minute (noradrenaline only). Our catecholamine data are similar to those of previous studies^{15,16} but the smaller changes observed in this study may be a reflection of the use of more sedation and more effective premedication than were employed in other comparable studies.

Tam *et al.*⁶ found that there was complete attenuation (defined as a lack of significant increase) in heart rate and arterial pressure changes when lignocaine 1.5 mg/kg was given 3 minutes before tracheal intubation. We found that increases in MAP were completely attenuated when lignocaine was given 4 minutes before intubation, but failed to show any effect in heart rate. This divergence in pressure and chronotropic responses has been noted elsewhere in

relation to deep anaesthesia¹ following administration of nitroprusside³ and also following administration of intravenous lignocaine.¹²

Table 3 summarises the results of previous studies which have examined the effect of intravenous lignocaine on the cardiovascular responses to tracheal intubation. An interval of less than 2 minutes was present between lignocaine administration and intubation in the majority of studies. Our data suggest that this interval may have been too short to permit any action of lignocaine and that this factor may be responsible for much of the conflict which exists in the literature on this topic. We conclude that intravenous lignocaine 1.5 mg/kg given at least 4 minutes before laryngoscopy may completely attenuate the pressor but not the chronotropic response to laryngoscopy and tracheal intubation. This suggests that intravenous lignocaine may not be the agent of choice for attenuating the sympathoadrenal responses to laryngoscopy and intubation in patients with myocardial ischaemia.

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Examination of cardiorespiratory changes during upper gastrointestinal endoscopy

Comparison of monitoring of arterial oxygen saturation, arterial pressure and the electrocardiogram

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Summary

Critical events including hypoxaemia, arrhythmias and myocardial ischaemia may occur more frequently during endoscopic procedures than during anaesthesia. A study was undertaken to assess the cardiovascular changes and to evaluate suitable monitoring techniques to detect critical events during sedation and endoscopy. Twenty patients scheduled to undergo a prolonged endoscopic procedure which required deep sedation were studied. Continuous recordings of electrocardiogram, heart rate and arterial oxygen saturation were made and arterial pressure was recorded at one-minute intervals. The study commenced immediately before administration of sedatives, continued for the duration of the examination and for one hour following the examination. Oxygen saturation decreased in all patients during the examination to a mean of 82.9% (SD 11.9), and remained below baseline for the duration of the examination and into the recovery period. Statistically significant increases and reductions of systolic arterial pressure and rate-pressure product were found during the procedures compared with baseline values recorded before administration of sedatives. Sixteen of the 20 patients developed tachycardia during the examination. Ten patients developed ectopic foci which were supraventricular, ventricular or both in origin. Electrocardiogram changes resolved during the recovery period. Myocardial ischaemia was assessed by S-T segment depression and a significant correlation was found between S-T segment depression and hypoxaemia, although the magnitude of the S-T depression was small and may not have been detected clinically. No correlation was found between S-T segment depression and arterial pressure, heart rate or rate-pressure product. Regular and frequent assessments of arterial pressure and heart rate are desirable but the results confirm the importance of monitoring arterial oxygen saturation in addition to arterial pressure and the electrocardiogram.

Key words

Surgery; gastrointestinal endoscopy.

Complications; hypoxaemia.

Monitoring; blood pressure, electrocardiography, pulse oximetry.

Critical events such as arterial hypoxaemia, arrhythmias and myocardial ischaemia must be detected rapidly to permit effective corrective action to be taken. Concern has been expressed regarding monitoring during gastrointestinal endoscopy¹ during which higher mortality rates have been reported than occur during anaesthesia.^{2,3} Studies have demonstrated that hypoxaemia and arrhythmias occur commonly during upper gastrointestinal endoscopy. Explanations for hypoxaemia include hypoventilation secondary to sedative medication, partial obstruction by the endoscope and possibly ventilation/perfusion mismatch caused by increased autonomic activity. Cardiac arrhythmias have been associated with these periods of hypoxaemia.

This study was designed to assess the frequency of cardiorespiratory changes during gastrointestinal endos-

copy and to establish the existence of any relationship between the measured variables during periods of myocardial ischaemia.

Methods

Twenty patients scheduled to undergo a prolonged or complex endoscopic procedure were recruited to the study. Written informed consent was obtained for data collection and storage on magnetic media, and the approval of the hospital ethics committee was given. The mean age of the patients was 62.5 years (SD 14.6, range 26–85) and the mean weight was 64.6 kg (SD 19.3, range 32–106). Fourteen of the 20 patients were male.

Seventeen patients had significant pre-existing medical conditions other than their gastrointestinal disease. These

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consisted of ischaemic heart disease (5), cerebrovascular disease (3), valvular heart disease (1), hepatic or pancreatic disease (4) and respiratory disease (4). The procedures performed were endoscopic retrograde cholangiopancreatography (9) and endoscopy for intended laser therapy (11). The mean duration of the examination was 20.7 minutes (SD 10.8, range 4–43). The study started immediately before administration of sedative drugs, and continued for the duration of the examination and for the first hour of the recovery period. The mean duration of the study was 80.7 minutes (range 64–103).

Lignocaine 5% was applied to each patient's pharynx using 10 doses of 10 mg from a metered spray. Sedative drugs were administered intravenously into a fast running infusion by the operator. Patients were given pethidine 50 mg if they were aged under 70 years, or pethidine 25 mg if they were older. The sedation was then completed with increments of midazolam until the patient's speech became slurred. The endoscope was introduced when the operator was satisfied with the quality of sedation. The mean dose of midazolam was 7.8 mg (SD 2.6, range 2.5–15).

Continuous electrocardiogram (ECG) monitoring was undertaken using a two-channel frequency-modulated recording system (Oxford Medical Medilog II system). The bipolar lead of most significance, CS5, was analysed in detail. The positive electrode was in the V5 position and the negative electrode was placed in the right infraclavicular fossa. ECG analysis was performed off-line by a computer linked to a Reynolds Pathfinder and provided data concerning heart rate, rhythm, presence and type of ectopic beats, and S-T segment amplitudes averaged over one-minute periods.

Arterial pressure was measured using either a Critikon Dinamap or a Datascope Accutorr 2 noninvasive monitor at one-minute intervals. Pulse rate and arterial oxygen saturation (SpO₂) were measured continuously by an Ohmeda Biox 3700 pulse oximeter. The blood pressure monitor and the pulse oximeter were both interfaced to an Atari 1040 ST microcomputer. The computer program was designed to capture data as they became available and store the measurements together with the time of recording on magnetic disc at one-minute intervals. The rate of data acquisition increased to 15-second intervals if the arterial oxygen saturation decreased by 5% or more below the patient's baseline value. During analysis the rate-pressure product was calculated by the computer for each set of one-minute recordings.

Results

Oxygen saturation decreased in all patients after administration of sedation and introduction of the endoscope, from a mean (SD) baseline of 94.4 (2.4)% to a minimum of 82.9 (11.9)% ($p < 0.01$). Saturation recovered to a mean of 91.6 (3.4)% by the end of the examination, a value which was significantly less than baseline ($p < 0.05$). A rapid, profound decrease in arterial saturation to 42% occurred in one patient who required manual ventilation of the lungs (Fig. 1).

The persistence of hypoxaemia was demonstrated beyond the end of the examination. The average minimum SpO₂ during the recovery phase was 90.1 (3.1)%, significantly less ($p < 0.05$) than mean saturation of 93.9 (2.4)% at the end of the study. One patient had a minimum

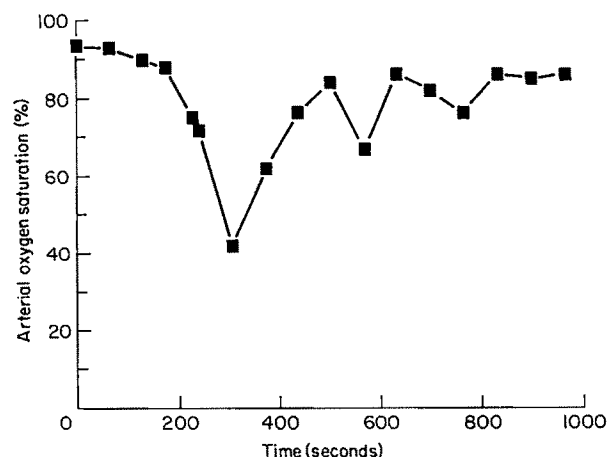


Fig. 1. Profound decrease in arterial oxygen saturation after insertion of endoscope in one patient.

saturation of 84% during recovery. There was no significant difference between arterial saturations at the beginning and at the end of the study.

During endoscopy, marked changes in systolic arterial pressure and heart rate occurred (Table 1). Significant hypertension and hypotension were found, with a large range of values. Significant increases in heart rate, and significant increases and decreases in rate-pressure product, also occurred during endoscopy.

ECG analysis showed that arrhythmias developed frequently during the procedure. Sixteen of the 20 patients developed sinus tachycardia and 10 developed supraventricular and (or) ventricular ectopic beats. All ECG changes resolved during recovery.

The development of myocardial ischaemia during endoscopy was evaluated by examination of the S-T segment depression in the ECG. The mean S-T segment amplitude at the baseline was -0.01 (0.30) mV and the mean minimum during endoscopy was -0.06 (0.23) mV. Linear regression was performed with data obtained at the lowest S-T segment values to examine the relationships between myocardial ischaemia and arterial pressure, heart rate, rate-pressure product or arterial oxygen saturation recorded at the time of maximum S-T depression. These analyses showed no correlation between S-T segment depression and arterial pressure ($r = 0.16$), heart rate ($r = -0.27$) or

Table 1. Blood pressure, heart rate and rate-pressure product changes during endoscopy.

	Mean (SD)	Range
<i>Blood pressure</i>		
Baseline	129.6 (20.4)	93–162
Maximum	146.2 (20.7)*	100–187
Minimum	93.9 (22.5)**	58–147
<i>Heart rate</i>		
Baseline	91.5 (19.5)	57–136
Maximum	118.4 (21.8)**	87–169
Minimum	81.5 (14.6)	51–109
<i>Rate-pressure product</i>		
Baseline	11 828 (3030)	7326–18 444
Maximum	15 980 (4338)**	8649–25 350
Minimum	8645 (2079)**	5270–12 450

* $p < 0.05$ from baseline.

** $p < 0.01$ from baseline.

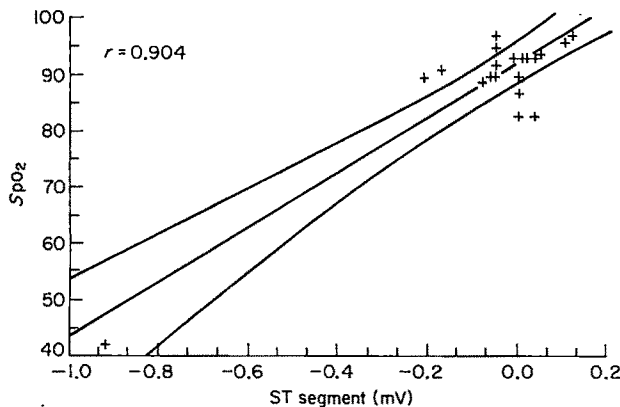


Fig. 2. Linear regression was performed with data obtained when S-T segments were at their lowest values for each patient. A significant correlation was found between saturation and S-T segments ($r = 0.90$). 95% confidence lines are shown.

rate-pressure product ($r = -0.09$). A significant correlation was found between the maximum S-T segment depression and saturation changes ($r = 0.90$, Fig. 2), but this relationship depended on the data point obtained from one patient. This patient had shown the largest reduction in arterial saturation. Data from each minute of the 5-minute period immediately preceding the maximum S-T depression were analysed and showed a similar relationship ($r = 0.98$, Fig. 3) between SpO_2 and S-T segment depression.

Discussion

Critical events may occur more frequently during endoscopic procedures than during anaesthesia. Fletcher² reviewed the records of 1800 patients who underwent sigmoidoscopy and reported three cases of sudden death during the examination. A further study by Katz³ reported one death in a series of 1200 gastroscopies, but Palmer *et al.*⁴ reported only five deaths related to 267 175 gastroscopies. Several studies have examined cardiovascular and respiratory changes resulting from sedation and insertion of an endoscope.

Hypoxaemia following administration of sedative drugs and introduction of the endoscope has also been assessed by several workers. The effects of diazepam alone or in combination with an opioid analgesic have been studied in healthy volunteers and in patients with chronic obstructive pulmonary disease.^{5,6} Diazepam alone caused no significant change in oxygen tension. Administration of pethidine resulted in a significant reduction in oxygen tension and an increase in carbon dioxide tension; the combination of diazepam and pethidine caused similar changes. This has been confirmed during endoscopy. Use of a narrow diameter endoscope has been shown to cause less hypoxaemia than a standard diameter endoscope.⁷⁻⁹ Intravenous midazolam has replaced diazepam in many units because of the potential advantages of a shorter half-life with rapid recovery, and lack of venous irritation and phlebitis. However, a recent report has demonstrated that a marked reduction in arterial oxygen saturation may occur following the administration of midazolam.¹⁰ Endoscopy without sedation also causes significant reductions in oxygen tension after introduction of the endoscope.^{7,11,12}

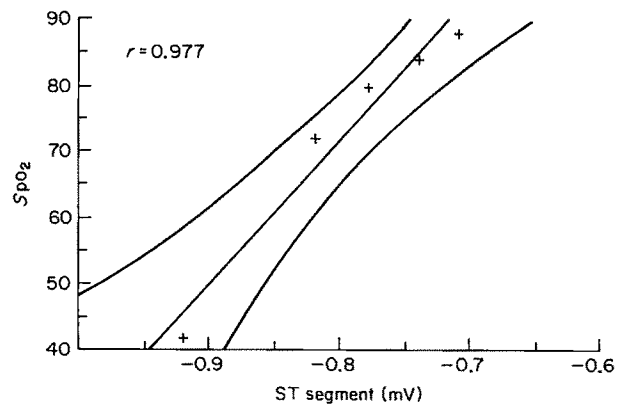


Fig. 3. One patient underwent marked arterial desaturation. Regression analysis was performed for arterial oxygen saturation and simultaneous S-T segment depression using data from the 5-minute period preceding and including the maximum S-T segment depression. A significant correlation was demonstrated between S-T segment depression and saturation ($r = 0.98$). 95% confidence lines are shown.

Hypoxaemia was assessed in most of these studies by intermittent sampling from an artery either via an indwelling cannula or by repeated puncture. Ear oximetry was used by two groups^{13,14} and provided a continuous trace of arterial oxygen saturation. Examination of the trace revealed transient reductions in saturation which could easily be missed by intermittent sampling. The ear oximeter has now been superseded by the pulse oximeter, which is simpler and cheaper. One study has demonstrated the value of pulse oximetry during upper gastrointestinal endoscopy; the author suggested that insufflation of oxygen via nasal cannulae may limit reductions in arterial oxygen saturation.¹⁵

Electrocardiographic studies show that rhythm changes and ectopic beats occur commonly after introduction of the endoscope and revert to normal soon after completion of the examination.¹⁶⁻¹⁸ The development or worsening of pre-existing S-T segment depression has also been noted during endoscopy¹⁶ but reverted to baseline during the recovery phase. It was noted that ECG changes were encountered more frequently in patients with ischaemic or valvular heart disease.^{7,17,18} A more recent study used rate-pressure product as an index of cardiovascular stress and concluded that stress during endoscopy was mild to moderate and that careful sedation could minimise the stress.¹⁹ This study also revealed ectopic beats and transient arrhythmias occurring during endoscopy.

Rostykus *et al.*¹³ demonstrated changes in ECG rhythm concurrent with desaturation using continuous oximetry and ECG measurement but no previous study has evaluated the extent of S-T segment depression or the relationship between S-T segment depression and other cardiorespiratory measurements. Unlike Sturges and Krone,¹⁹ we did not identify any relationship between rate-pressure product and myocardial ischaemia assessed by S-T segment depression, or between arterial pressure or heart rate and myocardial ischaemia. However, we did find a relationship between myocardial ischaemia and arterial oxygen saturation over a range of arterial saturations and degrees of S-T segment depression in one patient. In general, the extent of S-T segment changes found during this study was small and its detection during endoscopy or

anaesthesia may prove difficult. Pulse oximetry provides continuous information about the patient's oxygenation. The oximeter, via a signal strength indicator, provides a qualitative indication of pulsatile flow at capillary level implying that cardiac output is present. In addition, information from pulse oximeters is often presented as an auditory tone with pitch changes corresponding to alterations in oxygen saturation. Thus, pulse oximetry should be an appropriate monitor in any darkened environment such as that found during endoscopy, or during anaesthesia for radiological procedures.

The Association of Anaesthetists of Great Britain and Ireland has recently published guidelines which recommend that '...adequate monitoring is needed during brief anaesthetics or when using local anaesthetic or sedation techniques which may lead to loss of consciousness or to cardiovascular or respiratory complications'. In particular, they recommend that heart rate and arterial pressure are recorded regularly, and with a frequency relevant to the condition of the patient, and suggest that pulse oximetry should be used during short procedures such as dental treatment, endoscopy, cardioversion, and electroconvulsive therapy.²⁰ The results of our study suggest that while monitoring of blood pressure and ECG is worthwhile during critical events, continuous monitoring of arterial oxygen saturation may provide more valuable information.

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The effect of the Trendelenburg position on lower oesophageal sphincter tone

S. A. M. HEIJKE, G. SMITH AND A. KEY

Summary

Measurements were made of gastric, lower oesophageal and barrier pressures in the supine, moderate and steep Trendelenburg positions in 10 healthy female patients during balanced anaesthesia. Adoption of the Trendelenburg position resulted in no significant changes in any measurement, with the exception of a small increase (0.1 kPa) in intragastric pressure in the steep head-down position. We conclude that the steep Trendelenburg position should not predispose to regurgitation in healthy patients.

Key words

Gastrointestinal tract; lower oesophageal sphincter.
Position; Trendelenburg.

Regurgitation or vomiting and subsequent aspiration of gastric contents remains an important cause of anaesthetic-related morbidity and mortality. It is generally accepted that the major protective mechanism against regurgitation is the lower oesophageal sphincter.¹ This physiological sphincter may be defined manometrically although it is not anatomically distinct. The circular muscle constituting the sphincter can be affected by neuronal influences (chiefly vagal and sympathetic fibres) and numerous receptor-occupying substances of both endogenous and exogenous origin. Other mechanisms which may play a role in prevention of reflux include the flap-valve mechanism of the gastro-oesophageal angle, the flutter valve constituted by the length of intra-abdominal oesophagus and the pinch-cock action of the diaphragm around the oesophagus.

The tendency to regurgitation is opposed by the barrier pressure (BrP) which is calculated as the difference between lower oesophageal pressure (LOSP) and gastric pressure (GP).

Some anaesthetists have advocated induction of anaesthesia in a 20° head-up position for patients with a full stomach to reduce the possibility of reflux.² In contrast, the Trendelenburg (head-down) position may be preferred as gravitational drainage should prevent regurgitated fluid from entering the airway provided that it is not aspirated actively by forceful inspiration.³

It is generally assumed that the Trendelenburg position predisposes to regurgitation but the effect of this position on LOSP and BrP have not been examined.

Method

The study was approved by the District Ethics Committee and informed consent was obtained from each patient. We studied 10 healthy females scheduled to undergo elective gynaecological surgery which required muscle relaxation and tracheal intubation. All patients were healthy, receiving no medication, within 10% of expected body weight and with no history of reflux; where appropriate, they were asked to refrain from smoking for at least 8 hours before operation. Each patient was premedicated with diazepam 10 mg approximately 2 hours before the study.⁴ Intravenous thiopentone 4 mg/kg was used to induce anaesthesia and intravenous atracurium 0.4 mg/kg was given to facilitate tracheal intubation;⁵ the lungs were ventilated to normocapnia as determined by end-tidal carbon dioxide concentration. Anaesthesia was maintained with nitrous oxide 70% and enflurane 0–1% in oxygen.⁶ Fentanyl 1 µg/kg was given intravenously to supplement anaesthesia.

The oesophageal manometry system (Gaeltec Ltd, Isle of Skye, Scotland) (a 3-mm or gastric tube into which is embedded three radially sensitive subminiature strain gauge pressure transducers at 5, 10 and 15 cm from its tip), was calibrated in a column of water (scale 0–3 kPa) before each investigation. Calibration was repeated at the end of each study to exclude significant zero baseline or calibration drift (the system has been shown to remain stable for periods in excess of 90 minutes). The transducer was

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Table 1. Gastric pressure (GP), lower oesophageal pressure (LOSP) and barrier pressure (BrP) (kPa) in 10 patients in the supine position, and tilted 15° or 30° head-down. Data are presented as mean (SD).

	Supine	15° head-down	Supine	30° head-down
GP	0.78 (0.29)	0.86 (0.29)	0.79 (0.25)	0.91 (0.30)*
LOSP	3.26 (0.74)	3.04 (0.83)	3.25 (0.88)	3.46 (1.00)
BrP	2.48 (0.84)	2.19 (0.90)	2.48 (0.91)	2.54 (1.03)

*p < 0.05 compared with supine position.

attached via a pre-amplifier to a chart recorder to produce a permanent record of the pressures obtained.

When anaesthesia was stable, the orogastric tube was passed through the oesophagus until the transducers were in the stomach. It was then withdrawn slowly and sequential measurements made of GP, LOSP and oesophageal pressures at the end of expiration. This pull-through manoeuvre^{7,8} was repeated three times at approximately one-minute intervals in the supine (two separate baseline measurements), 15° and 30° of Trendelenburg position in each patient; the sequence of measurement was randomised between patients. Results were analysed statistically using Student's *t*-test for paired data.

Results

There were no significant changes in any measurement when patients were moved from the supine to the 15° Trendelenburg position. However, when the patients were tilted to the 30° head-down position, there was a significant increase in gastric pressure. As a result of a small but insignificant increase in LOSP, there was no significant change in BrP in this position (Table 1).

Discussion

We have found that although the steep Trendelenburg position caused a small but statistically significant increase in GP, there was a corresponding increase in LOSP with the result that BrP did not change significantly. We conclude therefore that in the healthy patient with a normal gastrointestinal tract, the adoption of the steep Trendelenburg position should not predispose to passive regurgitation. This view conflicts with accepted clinical teaching in anaesthesia that the combination of the Trendelenburg position with abdominal distension must predispose to passive regurgitation.⁹

Other investigators who have examined BrP have also observed that if GP increases there may be an increase in LOSP with no decrease in BrP and consequently no increased tendency to regurgitation. Lind *et al.*¹⁰ found that LOSP increased as a normal adaptive response to increased intra-abdominal pressure. Smith *et al.*¹¹ also demonstrated that BrP increased in response to suxamethonium-induced fasciculations in patients in whom there was a small increase in GP. Furthermore, Jones *et al.*⁸ demonstrated corresponding increases in LOSP and GP in response to the raised intraperitoneal pressure created during insufflation of the peritoneal cavity with carbon dioxide for pelvic laparoscopy.

It has been indicated that the adaptive reflex increase in BrP with raised GP may not operate in pregnancy. This is

illustrated in the study of Jones *et al.*¹² who showed that in the first trimester of pregnancy, whilst BrP was within the normal range of values in the supine position, there was a significant reduction when the patients were placed in the lithotomy position, a manoeuvre which gave rise to a small increase in GP.

There are several older studies which have examined only GP in response to changes in body position. The Trendelenburg position was found to result in relatively small increases in GP in males, pregnant and nonpregnant females and children, but the authors concluded that the magnitude of increase was not sufficient to represent a significant risk to regurgitation.¹³ In 1954, O'Mullane¹⁴ demonstrated a small increase in GP in the Trendelenburg position in patients with a nondistended abdomen in awake, lightly or deeply anaesthetised, or paralysed conditions. However, these changes did not occur in patients with a distended abdomen.

Studies of reflux produced by variation in body position have also failed to demonstrate an increase in the degree of reflux caused by positions which may be assumed to increase GP. Stanciu *et al.*^{15,16} found no significant difference in the number of reflux episodes in susceptible patients adopting the supine from the sitting position, although acid clearing was faster in the sitting position. They examined the Trendelenburg position and found that the incidence of episodes of reflux did not change, but did not report pressure changes in the lower oesophageal sphincter or stomach.

There may be other physiological changes which are also likely to reduce the probability of regurgitation in addition to physiological changes produced by an adaptive increase in lower oesophageal sphincter tone on adopting the Trendelenburg position. Greenan *et al.*¹⁷ suggested that when the angle of the stomach, relative to the oesophagus, became more acute, a greater increase in GP was required to produce gastro-oesophageal incompetence. This would also reduce the likelihood of regurgitation if, as seems probable, the gastro-oesophageal angle becomes more acute in the Trendelenburg position.

It would seem reasonable to conclude therefore that in normal patients under balanced anaesthesia, with an empty stomach and without respiratory tract obstruction, the Trendelenburg position should not predispose to gastro-oesophageal regurgitation.

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Introducing an acute pain service

P. D. CARTWRIGHT, R. G. HELFINGER, J. J. HOWELL AND K. K. SIEPMANN

Summary

An acute pain service in a new district general hospital is described. We have reported the incidence of severe pain, common postoperative anaesthetic problems and patient satisfaction in relation to the analgesic technique. Over half the patients were treated by intermittent intramuscular injection of opioid, but increase in the use of continuous intravenous therapy and in particular patient-controlled analgesia, was welcomed by both medical and nursing staff.

Key words

Pain; postoperative, prevention.

'One reason for the inadequacy of conventional opioid analgesia is that it is prescribed by members of one profession and administered (or withheld) by members of another, to patients who may be too ill or inhibited to demand it.'¹

There is a need to improve the management of acute pain in hospital patients, especially postoperative pain. Surveys have shown that moderate to severe pain is experienced after surgery in 30–75% of patients.^{2–4} One approach to improve the situation is to use existing, well-tried drugs and techniques and, after operation, to follow up and monitor patient comfort closely. This may appear a simple, logical step, but it is fraught with difficulties, prejudices, practical constraints, communication problems, and involves hard work.

This paper describes the introduction of an acute pain service (APS) in a new district general hospital, with the aim of establishing the service before traditional and often ineffective methods had become ingrained.

Methods

Organisation

The organisation of the acute pain service was the responsibility of named anaesthetic consultants. A senior and junior doctor were available day and night and had no other commitment than emergencies.

A simple, single sheet which describes the aims and methods of the APS was distributed to all wards, ward sisters and housemen, and has been followed up by individual tuition of nurses on the wards, regular meetings with

the sisters, and demonstrations of equipment (PCA and other syringe pumps). The sheet also lists who to contact for help with acute pain problems, and describes safeguards and precautions.

New surgical house officers were seen on induction into a new post, then every 6 months, to inform them of hospital policy, and to influence their normal practice of prescribing fixed *pro re nata* (PRN) doses of analgesics on admission. The most important aspect of this educational process was the instruction of night staff and hospital coordinating sisters in order to re-inforce the aims of APS and the monitoring of postoperative patients, and to ensure that they were fully conversant with all the equipment. Further education was made available in the school of nursing.

Before surgery

All surgical patients received an A5 leaflet on admission entitled 'After your operation'.⁷ This explains what they should expect, and advises about simple practical methods to reduce discomfort after operation: for example, to hold pillows over the abdomen. It states that the patient will be visited by the anaesthetist after operation, and gives information about the type of pain relief that may be used. During the pre-operative anaesthetic visit, it is decided whether the patient will receive PCA, and if so, the pump is demonstrated and a further information sheet given out.

Postoperative recovery

In the recovery room each anaesthetist decides on the postoperative analgesia on the basis of opioid requirements

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during surgery and in the immediate postoperative period. Patients are retained for at least 30 minutes after operation. In this way the initial rate of continuous intravenous infusion, or the PCA programme, is determined before transfer to the ward. For simplicity, opioids for continuous intravenous infusion are all diluted to 1 mg/kg.

Immediate care

On the evening after surgery, and on each evening thereafter, the patients indicated by the follow-up procedure are seen by the on-call anaesthetic team. If necessary, the analgesic regimen is revised.

Follow-up

A senior and junior anaesthetist make a postoperative round every morning. The patients to be visited are indicated by forms retained in the recovery ward. A post-operative assessment is made and the form completed (Fig. 1) or returned to the recovery ward to prompt further assessment. Usually this applies only to patients receiving further intravenous opioids.

Audit and documentation

Figure 1, an example of the combined anaesthetic complication and analgesia assessment form, is on the reverse side of the anaesthesia record and is a complete account of the peri-operative period. Pain was measured using a verbal rating score and the doses of the analgesic drugs were recorded. The forms are used for anaesthetic and APS audit.

Results

The use of drugs for postoperative analgesia for our first 1600 inpatients is shown in Table 1. Traditional intramuscular analgesia accounts for 51.6%, although there was a progressive increase in intravenous opioid use during the months that the APS was functioning and these techniques were more accepted on the wards. Regular intramuscular analgesia was given in 16% of intramuscular cases and PRN in 84%. A further analysis of the intravenous and intramuscular doses of papaveretum, the commonest opioid used, is given in Table 2. This confirms the suspicion that intramuscular opioid prescriptions are poorly complied with, since 80% of patients received only 0, one or two doses.

Regional analgesic techniques are rarely used (6.1%). The 36 cases included for spinal analgesia refer exclusively to urological procedures in which further pain relief was not required. The 21 cases of epidural analgesia were major thoracic and abdominal procedures, which were managed initially in the intensive care unit.

One hundred and seventy-five patients were not assessed because of early discharge. Day care patients, who received a postal questionnaire, showed an 81% patient satisfaction with the oral analgesic prescribed (61% response).

Table 3 gives the incidence of common problems such as severe pain and patient dissatisfaction after anaesthesia. One hundred and seventeen patients had more than one assessment, i.e. were seen on the second or third post-operative day. Many reports of severe nausea and vomiting occurred in patients not receiving any opioids after operation and six cases reported as oversedation were not receiving any opioids. Of the 20 cases reporting overseda-

Problems with anaesthetic			Analgesic drug			Cumulative dose after operation (hours)		
Intravenous site	None	Haematoma	Inflammation	PCA	Increased dose	mg	mg (...tries)	mg (...tries)
Dreams	None	Pleasant	Unpleasant		Background infusion	mg/hour		
Headache	None	Mild	Severe	Syringe pump	Range	mg/hour	mg	mg
Sore throat	None	Mild	Severe		Dose used	mg/hour		
Nausea/Vomiting	None	Mild	Severe	Intra-muscular bolus	Dose	mg	mg	mg
Oversedation	None	Mild	Required therapy		Frequency	Hourly Regular/PRN mg		
Other:				Oral	Dose	mg	mg	mg
					Frequency	Hourly Regular/PRN		
Overall pain on movement	Effect of analgesic		Regional block	Site	mg ml %	mg ml	mg ml	
0=no pain 1=mild pain 2=moderate pain 3=severe pain	0=better than expected 1=as expected 2=worse than expected			Dose < Frequency	Hourly			

Comments:

Fig. 1. Postoperative assessment form.

Table 1. Distribution of postoperative pain prescriptions.

Analgesic method	Number of cases	Drug used	
PCA	66 (4.6%)	Morphine	53
		Papaveretum	11
		Pethidine	2
Intravenous pump	179 (12.6%)	Morphine	25
		Papaveretum	149
		Pethidine	5
Intramuscular bolus	735 (51.6%)	Morphine	52
		Papaveretum	593
		Pethidine	90
Oral	227 (15.9%)	Codydramol	156
		Paracetamol	64
		Diclofenac	5
		Buprenorphine	1
		Ibuprofen	1
Regional block	87 (6.1%)	Epidural	21
		Caudal	12
		Spinal	36
		IVRA	4
		Plexus	3
		Local infiltration	11
None prescribed	131 (9.2%)		
Not assessed	175		
Total	1600		

Table 2. Intravenous and intramuscular administration of papaveretum.

Intravenous infusion; mg/hour	1	2	3	4	5	6	
Number of cases (total 149)	18	51	49	21	7	3	
Intramuscular bolus (number of doses given)	0	1	2	3	4	5	6
Number of cases (total 593)	167	188	117	79	31	4	7

Table 3. Postoperative assessment.

	Number of cases	(%)
Headache (severe)	56	4.0
Sore throat (severe)	9	6.4
Nausea/vomiting (severe)	111	7.8
Oversedation	20	1.4
Dreams — pleasant	11	0.8
— unpleasant	4	0.3
Intravenous problems — haematoma	17	1.2
— inflammation	12	0.8
Severe pain	142	10.0
Worse than expected	114	8.0
Assessments made 1	1308	91.8
2	96	6.7
3	21	1.5

tion, only two required therapy. Both occurred in the recovery room.

The 10% incidence of severe pain may be a considerable underestimate due to inconsistent methods in the reporting. Most of the 142 patients who experienced severe pain were prescribed intramuscular analgesia PRN, but there were some patients from all treatment groups who indicated the need for improved prescribing techniques.

Discussion

The introduction of an acute pain service may be considered as a series of hurdles.

First hurdle

The anaesthetist prescribes postoperative analgesia. For an improvement in the APS the whole anaesthetic department must agree about the techniques and drugs to be used and the worth of the whole enterprise. From the outset, a consistent approach should be seen to be followed by other clinical staff (nurses, house officers etc.) and applied to all surgical patients and not just 'extra care' to a selected few. It must be accepted that analgesic requirements vary enormously between patients and that doses may subsequently be altered by members of the acute pain team.

We have been fortunate, as a new department, in that the introduction of an acute pain service was agreed from the outset. The analgesic techniques are not standardised, and are entirely at the discretion of each anaesthetist, tailored to the individual patient.

Second hurdle

If education, communication and persuasion are necessary for postoperative pain prevention to become a reality, nursing staff must be equally convinced that good analgesia is important. For an acute pain service to function, it is fundamental that nursing staff, surgical staff and anaesthetists work as a team towards the same goal.

Third hurdle

The most difficult barrier to the introduction of a comprehensive, smooth-running service encompasses the mundane practicalities of what ward nursing staff are or are not allowed to do under various health district nursing policies, and also what one can realistically expect from members of the anaesthetic department, both junior and senior, with respect to regular follow-up of patients.

There is a wide discrepancy between what are classified normal nursing duties, and procedures that require an 'extended role certificate' between health districts, despite all being under the auspices of a common National Health Service and United Kingdom Central Council for Nursing, Midwifery and Health Visiting. The variations are mostly due to local protocols and traditions peculiar to the particular health district. In a new hospital, nurses may be appointed from throughout the country, and while many are quite happy to operate syringe pumps, others consider it to be an 'extended role'.

This hurdle was overcome by the adoption of a 'standard nursing procedure' for the addition of drugs throughout the hospital to syringes, burettes etc., although the addition of intravenous boluses was strictly an extended nursing duty. This enabled us to introduce opioid infusions to the general surgical wards without the need for medical staff to be responsible for recharging syringes. We are, unfortunately, a long way from the situation in Seattle⁸ where ward nurses are allowed to administer epidural narcotics and to reprogramme PCA pumps under strict protocols. We have started simply in developing the APS, concentrating initially on introducing intravenous opioid techniques. We have no high dependency unit and patients with epidural

opioids or local anaesthetic infusions are cared for on the intensive care unit.

We persuaded the nursing managers that the prescribed variable infusion rate was well within the ability of ward nurses to manage. It was no different in principle to a variable 3–6 hourly PRN intramuscular regimen, or variable insulin infusion. Although this was accepted as a normal nurse procedure, it has involved an enormous amount of personal communication and tuition to all ward staff as to what they are and are not allowed to do. Initial antagonism and scepticism in the use of syringe pumps and PCA pumps has resolved as the ward nurses realised how much simpler, effective, and time saving these methods are, as has been previously described.^{5–7}

The APS has proved educational for both junior and senior anaesthetists. A comprehensive postoperative follow-up shows which operations require little or no postoperative analgesia, for example, varicose vein, hernia repair, urological, thyroid and breast surgery, and has resulted in a change in prescribing habits. The audit has also confirmed that there is a poor compliance rate with PRN analgesia. Patients are often reluctant to admit to suffering pain because of fear of injections, social attitudes, or dislike of the side effects of opioids. The number of patients suffering from severe postoperative pain has decreased, but this is most likely an underestimate and we are trying to standardise the assessment technique specifically to represent pain on moving (sitting up, coughing etc.) as being more representative of overall patient discomfort.

The presence of anaesthetists doing postoperative ward rounds has had an indirect beneficial effect for the anaesthetic department in fulfilling a more clinical role and in raising the level of importance of pain prevention in the minds of all clinical staff.⁹ There has been no resistance at all from surgeons to the introduction of an acute pain service, only appreciation and encouragement. There is also an increased referral rate by surgeons and physicians for

other pain problems, and there have also been requests by nursing staff to change prescribed analgesia for patients with pain problems. However, we are in the early days of the APS, but we feel that this provides a good framework for the audit of both anaesthetic complications and postoperative pain.

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General anaesthesia and undrained pneumothorax

The use of a computer-controlled propofol infusion

S. L. CROFTS AND G. L. HUTCHISON

Summary

A patient who required pleurectomy had a 30% pneumothorax when she was presented for anaesthesia. She had refused to have this drained, and it had not responded to conservative management. She was anaesthetised using a computer-controlled propofol infusion system, without the use of nitrous oxide, and a chest drain was inserted before the institution of positive pressure ventilation. This technique reduces the hazards associated with general anaesthesia in the presence of an undrained pneumothorax. It may be a safe alternative method of induction of anaesthesia in other conditions in which positive pressure ventilation must be avoided, such as bronchopleural fistula.

Key words

Anaesthetics, intravenous; propofol.
Complications; pneumothorax.

Case history

The patient, a 16-year-old female weighing 59 kg, had suffered three spontaneous left pneumothoraces in the previous 6 months. She was otherwise fit and well, receiving no medication, and had no history of other illnesses apart from childhood exanthemata.

When she presented with her fourth left pneumothorax, she was referred for surgical management, and scheduled for a left thoracotomy and pleurectomy. She had an approximately 30% pneumothorax on admission. Two days later at the anaesthetist's pre-operative visit this had not resolved to any significant extent, despite the administration of high-flow oxygen through a facemask for the preceding 24 hours. The pneumothorax persisted but she was not dyspnoeic. Arterial blood gas analysis showed a PaO_2 of 12.4 kPa and a PaCO_2 of 4.2 kPa.

The patient had previously had a particularly unpleasant experience during the insertion of a chest drain. She refused to consider having the procedure while she was conscious, and said that she would discharge herself from hospital if this was insisted upon. We therefore decided to anaesthetise her before the insertion of a small-bore chest drain, and then proceed with surgery. This plan was explained to the patient.

She was premedicated with temazepam 20 mg orally, 2 hours before surgery. Oxygen was administered at 5

litres/minute through a Hudson mask on arrival in the anaesthetic room, and a 14-G intravenous cannula was inserted in the back of the left hand. The patient was connected to an electrocardiogram, a noninvasive blood pressure monitor and a pulse oximeter. Oxygen saturation was 99% on mask oxygen. Equipment for chest drainage was at hand.

Fentanyl was administered in increments to a total of 100 μg , and the cannula was connected to a computer-controlled propofol infusion pump.

This device consists of an Ohmeda 9000 pump connected to a Psion Organiser hand-held computer by a custom-built back bar. The system is a further development, by the same team, of the Atari-controlled propofol pump already described by White and Kenny.¹ The computer program uses a three-compartment mathematical model which describes the pharmacokinetics of propofol. The patient's weight and the target blood concentration are input by the anaesthetist and the infusion is started. The pump runs at an initial rate of 1200 ml/hour until the computer predicts that the desired blood concentration has been achieved. The infusion rate is then reduced to a level designed to compensate for elimination and redistribution to peripheral compartments. Frequent recalculation by the computer results in constant tailoring of the infusion rate to maintain the target blood concentration. The blood level can be increased (by a further computer-controlled bolus) or

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decreased (the pump switches off until the new concentration is reached by a process of redistribution and elimination). The anaesthetist only needs to enter the new target concentration and the computer directs the pump accordingly. White and Kenny found that, using this system, there was a strong statistical relationship between the measured blood propofol concentrations and the corresponding computer predictions.¹

An initial target propofol concentration of 3 µg/ml was selected and the infusion started. A minute after the computer model had reached this target, the patient was still making some efforts at speech and movement. The target concentration was reset to 4 µg/ml and shortly afterwards her eyelash reflex disappeared. The initial skin incision resulted in a small amount of movement from the patient. Blood levels were reset to 5 µg/ml and once the computer predicted that this level had been achieved another attempt was made. On this occasion, no movement occurred and a 20-G chest drain was inserted without any significant change in heart rate or blood pressure. The patient breathed spontaneously throughout the procedure and the oxygen saturation was constant at 99%.

When the drain had been secured, gentle attempts at manual ventilation did not result in any large air leak from the drain. Vecuronium 7 mg was administered, the lungs were manually ventilated until the onset of neuromuscular blockade and a medium left Robertshaw double-lumen tube was inserted. The propofol infusion was then stopped and anaesthesia continued with 33% oxygen and 1% enflurane in nitrous oxide. The anaesthetists did not consider themselves sufficiently experienced in the use of this system to continue using it in a paralysed patient.

A left internal jugular central venous pressure catheter and a left radial arterial line were inserted and used for monitoring. An end-tidal carbon dioxide analyser was connected to the right lumen of the Robertshaw tube. Intermittent boluses of papaveretum were given and a subcutaneous diamorphine infusion, for postoperative pain relief was started during the operation. Surgery was uneventful, and at the end the left lung was easily inflated without major air leaks. Residual neuromuscular blockade was reversed and the tracheal tube was removed once adequate spontaneous ventilation was restored.

The diamorphine infusion was continued for 48 hours, and the patient made an uneventful recovery.

Discussion

The problems associated with general anaesthesia in the presence of an undrained pneumothorax are well known. The pneumothorax may expand because of the diffusion of nitrous oxide into the air-filled pleural space or as a result of the increased pressure associated with intermittent positive pressure ventilation (IPPV).

In 1955, Hunter reported a series of general anaesthetics, using nitrous oxide, in patients who had artificial pneumothoraces for the treatment of tuberculosis.² He described the gradual deterioration of these patients as anaesthesia progressed, with the classical signs of tension pneumothorax: increasing tachycardia, venous congestion, hypotension and inadequate ventilation. He demonstrated that the intrapleural pressure was elevated and by analysing gas samples taken from the pneumothorax, showed the increasing nitrous oxide concentration.

In 1965, Eger and Saidman demonstrated further the effects of diffusion of nitrous oxide into air-filled cavities.³ In dogs anaesthetised with 70–80% nitrous oxide, a 300-ml pneumothorax doubled in size in 10 minutes and quadrupled in size within 2 hours.

IPPV may precipitate a pneumothorax if a partial thickness tear of the visceral pleura exists, or may rapidly exacerbate a pre-existing pneumothorax which communicates with lung air through the pleura.⁴

Pneumothoraces have developed, presumably by this mechanism, during anaesthesia in patients in whom subclavian venous catheters were previously inserted.^{5,6} There was no evidence of intrapleural air on chest X rays taken after the catheter insertion. A 'late' chest X ray is advocated for all such patients before general anaesthesia.⁷ Another report describes the onset of tension pneumothorax during IPPV in paediatric patients with a known previous air leak.⁸

It is important to avoid positive pressure ventilation and the use of nitrous oxide in the presence of an undrained pneumothorax or in high risk patients. If a pneumothorax is present before surgery it should be treated with an indwelling chest drain before the induction of general anaesthesia.⁹

Our patient would not accept the insertion of a chest drain while awake. It would have been possible to induce anaesthesia and insert a drain immediately afterwards, probably without detriment to the patient, but the availability of the computer-controlled propofol infusion offered a safer alternative.

The computer-controlled pump allows the predicted blood propofol concentration to be achieved with little or no overshoot. There is less danger of transient respiratory depression due to a bolus effect and the need for positive pressure ventilatory assistance is therefore reduced. In this case, the additional precaution was taken of deepening anaesthesia in small increments until the required level was reached.

We conclude that such infusion systems can be used to provide a safe alternative method of anaesthesia when IPPV and/or nitrous oxide must be avoided, provided that the pharmacokinetic model in use can be applied to the patient being anaesthetised. In patients with bronchopleural fistula, for example, gaseous induction of anaesthesia is frequently advocated. In practice this may be difficult to achieve, however, because of poor gas exchange and airway irritability. In such cases, the intravenous route may be used safely for induction of anaesthesia if a computer-controlled propofol infusion is given using the technique described here.

Acknowledgments

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Computerised axial tomography of the trachea

A useful investigation when a retrosternal goitre causes symptomatic tracheal compression

P. BARKER, R. A. MASON AND M. H. THORPE

Summary

Case histories are reported of three patients who had large retrosternal goitres which were responsible for significant abnormalities of the airway. Computerised axial tomography demonstrated the exact anatomy. The site of tracheal compression was shown and accurate measurements of the diameter of the trachea at its narrowest point were made. This information was useful when the management of the patient was planned.

Key words

Surgery; thyroidectomy.

Measurement techniques; computer assisted tomography.

Marked tracheal compression may occasionally be produced by large retrosternal thyroid goitres. Plain X rays do not always show the airway particularly well and sometimes they overestimate the tracheal dimensions. Computerised axial tomography (CT) of the airway may provide additional information in patients who have symptoms which arouse suspicion of tracheal compression.

Case histories

Patient 1

A 53-year-old man with a large right-sided thyroid goitre was admitted for thyroidectomy. There was no recent increase in the size of the mass, but over the 3 months before admission he had noticed inspiratory stridor when supine and increasing breathlessness during exercise. He was a large plethoric man, with a thyroid swelling 10 cm by 6 cm on the left and 4 cm by 4 cm on the right. There were no other abnormal findings on clinical examination. He was euthyroid.

X ray of the chest and thoracic inlet (Fig. 1) showed widening of the superior mediastinum, displacement of the trachea to the right and tracheal compression in the transverse diameter at the level of the second thoracic vertebra. This was estimated to be 11 mm on plain X ray. We decided in view of the patient's symptoms to define the anatomy of the tracheal narrowing more accurately with a CT scan. This showed that the minimum diameter was

actually only 8.4 mm compared with a diameter of 2.23 cm just above the carina (Fig. 2).

The patient smoked 50 cigarettes a day. Bronchoscopy was performed under local analgesia a week before surgery. The purpose of this was to exclude other pathology, to assess the nature of the constriction and estimate its distance from the teeth. The narrow section was found to be soft, and no other pathology was evident at bronchoscopy.

The patient was premedicated for thyroidectomy with temazepam 20 mg and glycopyrronium 0.4 mg. Anaesthesia was induced with halothane, nitrous oxide and oxygen, while the electrocardiogram and oxygen saturation were monitored. When it was found that ventilation of the lungs could be performed easily, suxamethonium 100 mg was given. A 7.5-mm Mallinckrodt armoured tube was introduced so that it extended below the lower limit of the tracheal narrowing. Auscultation confirmed ventilation of both lungs. The tracheal tube was removed with the patient awake after uneventful surgery.

Patient 2

A 46-year-old man with a large right-sided goitre was admitted for thyroidectomy. He had some difficulty in swallowing and slight hoarseness of his voice. He also had a hiatus hernia. Chest X ray showed that there was retrosternal extension of the thyroid mass, deviation of the

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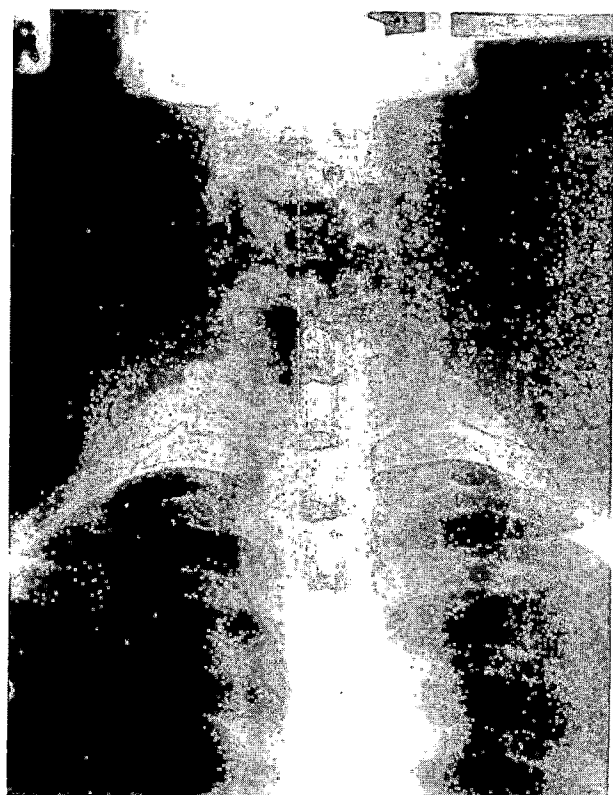


Fig. 1. Plain X ray of patient 1. This X ray showed tracheal displacement to the right and tracheal compression in the transverse diameter, which was estimated to be 11 mm on this film.



Fig. 2. CT scan of patient 1. This confirmed the tracheal deviation and allowed accurate measurement of the tracheal diameter (x-1-x). This was 8.4 mm.

trachea to the left and some tracheal compression. A CT scan was done. It showed a huge vascular mass producing marked deviation of the larynx and hypopharynx to the left. The trachea was found to be compressed, but not to a significant degree. In view of this reassurance, and the fact that the patient had a hiatus hernia, a rapid sequence



Fig. 3.

induction was undertaken. The marked laryngeal deviation was confirmed at laryngoscopy.

Patient 3

An 82-year-old woman was admitted as an emergency with marked stridor and a huge thyroid swelling. She was unable to lie flat and required oxygen supplements to maintain a satisfactory oxygen saturation.

A large nontoxic multinodular goitre had been present for many years but the patient had always refused surgery, despite increasing symptoms as a result of retrosternal extension. She had a right vocal cord palsy.

It was clear that the combination of increasing goitre size and an acute chest infection had precipitated the respiratory distress. The infection was treated with antibiotics, physiotherapy and steroids. A needle biopsy was performed. Histology showed some macrophages and colloid, but the specimen was highly cellular with nuclear pleomorphism and therefore a follicular or papillary carcinoma could not be excluded. The patient was then presented to the anaesthetist as a possible candidate for surgery. The CT scan (Fig. 3) provided valuable information. The trachea was encircled by a large goitre; the narrowest part of the trachea, at the level of the sternal notch, was only 4.6 mm in anteroposterior diameter (Fig. 3a). The minimum transverse diameter of 6.1 mm was just above this level. There was 6.35 cm of trachea of more normal calibre above the carina (Fig. 3b).

The constriction at the thoracic inlet was severe and would not accommodate a tracheal tube of sufficient diameter unless dilatation were possible. However, the tomogram showed that there was an adequate length of more normal trachea above the carina to accommodate the distal end of a tracheal tube. Safe intubation would depend upon the ability of the stenosis to dilate to accommodate a tube of adequate internal diameter. The use of cardiopulmonary bypass was not considered appropriate in this elderly woman.

It was agreed in view of the obvious difficulties, the age of the patient and the concurrent development of acute cardiac failure, that she should be treated conservatively with radioactive iodine. There is evidence that radioactive iodine can achieve a substantial reduction in the size of a multinodular nontoxic goitre without producing initial swelling.¹ This proved to be so. The respiratory symptoms improved and the patient was discharged from hospital. She will, however, be kept under review.

Discussion

Thyroid enlargement often results in tracheal deviation, particularly when there is retrosternal extension of the goitre, but significant tracheal narrowing is relatively uncommon. Thus in most cases, a plain X ray of the thoracic inlet provides sufficient information to guide the anaesthetist.

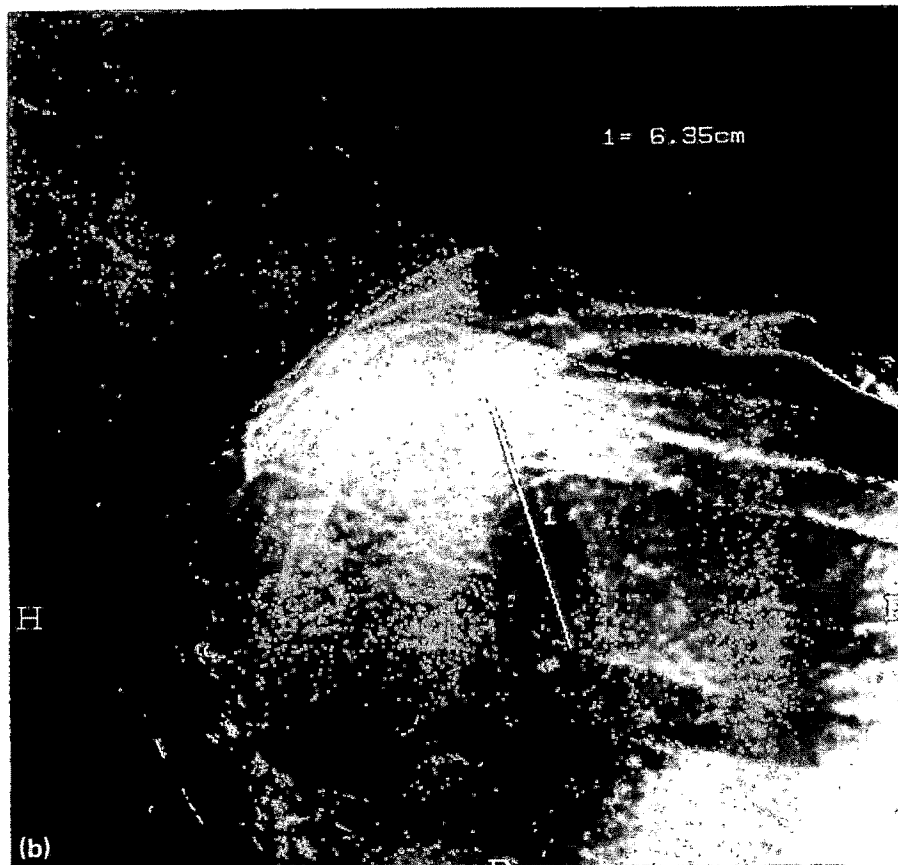


Fig. 3. CT scan of patient 3. This showed severe tracheal compression at the level of the sternal notch. (a) The tracheal diameter has been measured at three different levels, 1, 2 and 3. The minimum anteroposterior diameter (2) was 4.6 mm. (b) shows that there was 6.35 cm of trachea of more normal calibre (1) above the carina.

However, if the tracheal diameter is reduced by 50% or more, the patient usually develops symptoms. Clinical features may include breathlessness or stridor on exercise, positional respiratory difficulty (as in case 1), or stridor at rest (as in case 3). If anaesthesia is contemplated in these circumstances, a plain X ray may not give sufficient details of the anatomical abnormalities. A CT scan may be needed to provide further information before the management is planned.

A study of CT scans of the neck in adults² showed that the average (SD) anteroposterior diameter of the trachea at the first tracheal ring was 2.08 (0.34) cm and the transverse diameter was 1.84 (0.23) cm. These values correlate well with those found in a cadaveric study in men,³ where the mean sagittal diameter from the first to the tenth tracheal rings was 1.8–2.2 cm and the mean mid-coronal diameter was 1.7–2.1 cm.

Measurements taken from plain X rays alone are consistently greater than this due to a magnification effect and because of difficulties in defining the margin of the tracheal lumen. In a study of plain X rays in 10 patients, Dekker and Ledebor⁴ estimated the mean anteroposterior diameter to be 2.6 cm. This measurement is significantly greater than those taken from CT scan and cadaveric studies. Our experience with the first patient was similar; measurement taken from the plain X ray overestimated the diameter of the trachea at its narrowest part by 2.6 mm.

Computerised axial tomography permits accurate cross-sectional positioning of axial slices using gantry angulation, precise visualisation of the tracheal lumen and exact measurement without magnification. It may be useful in the assessment of tracheal diameter before surgery in the presence of other masses in the neck or mediastinum. Invasion of the tracheal wall by carcinoma can be detected by this technique. It may also be used for evaluation of tracheal stenosis at the site of previous tracheostomy.⁵

The CT scan provided accurate information in the first patient to allow an appropriate size of tracheal tube to be

selected and its optimum length determined, so that the constriction was passed but bronchial intubation was avoided.

The presence of a hiatus hernia in the second patient made a rapid sequence induction desirable. It was therefore useful to know the abnormal position of the larynx in advance, and to be confident that no significant tracheal narrowing was present. The CT scan clearly demonstrated the severity and position of the compression and led to the adoption of a conservative method of treatment for the third patient. We believe that this avoided a particularly hazardous anaesthetic for this elderly, ill woman.

Acknowledgments

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CASE REPORT

Posthypoxic myoclonus (the Lance-Adams syndrome) in the intensive care unit

S. J. HARPER AND R. G. WILKES

Summary

The neurological assessment of patients admitted to the intensive care unit after successful resuscitation from cardiopulmonary arrest may be difficult. We describe the cases of two patients who developed myoclonus within 24 hours of hypoxic respiratory and cardiac arrest. Initially, the clonic movements were thought to be generalised convulsions and were treated as such, until it became evident that the patients were aware and distressed. Posthypoxic myoclonus is a rare complication of successful cardiopulmonary resuscitation. Recognition depends on the awareness that the syndrome exists, and is important so that correct therapy can be instituted. There may be important prognostic implications. Both our patients had normal intellectual recovery with moderate residual neurological disability from their movement disorder.

Key words

Complications; resuscitation, myoclonus.

The patient who remains comatose after successful resuscitation from cardiopulmonary arrest presents difficult management decisions, not least because the likelihood of survival, and of survival with functional neurological recovery is unpredictable.^{1–4} The longer that such patients remain comatose, the poorer are the prospects for a good outcome.^{3,4} The chances of functional neurological recovery decrease substantially if coma is complicated by grand mal convulsions, especially if these occur more than 4 hours after the cardiac arrest³ and last for 30 minutes or more.⁵

After successful cardiopulmonary resuscitation, the development of posthypoxic myoclonic jerks (that is, rhythmic contractions of peripheral musculature) has been reported.^{6,7} The myoclonus may be confined to muscles supplied by a localised segment of spinal cord or brainstem, or be more generalised.⁵ Generalised myoclonus is uncommon in the intensive care unit and may therefore be easily confused with posthypoxic seizures. It is important that the distinction is made early, however, since mistaken diagnosis may lead to a delay in the institution of appropriate therapy, and possibly to mistaken conclusions about the quality of neurological recovery. Although the presence of myoclonus *per se* has not been shown to be related to outcome, the co-existence of myoclonus and status epilepticus is predictive of poor outcome.⁵

We describe two cases of patients admitted to our intensive care unit in coma after successful cardiopulmonary resuscitation. In both cases, several myoclonic jerks developed within 12 hours of admission and were initially thought to be grand mal convulsions. Both patients survived to be discharged from hospital, recovered their precardiac arrest intellectual status and were capable of caring for themselves in the community with assistance. It is probable that posthypoxic myoclonus will be recognised more frequently as improved techniques for cardiopulmonary resuscitation and protocols for continued care are instituted.

Case histories

Patient 1

A 47-year-old man with a long history of asthma, excessive alcohol use without liver disease and with a known allergy to mustard inadvertently ate mustard on the evening of admission, which led to his tongue swelling and profound bronchospasm. He had a respiratory arrest in the ambulance on the way to hospital and on arrival in the accident and emergency department was pulseless, unconscious and cyanosed. Cardiopulmonary resuscitation was started. A tracheal tube was inserted and artificial ventilation

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commenced. The patient was given intravenous adrenaline, aminophylline, salbutamol and hydrocortisone and transferred to the intensive care unit without regaining consciousness. Over the following 8 hours, the patient's condition improved and he was weaned from mechanical ventilatory support and extubated. Within minutes of extubation, the patient started to convulse with clonic movements of all four limbs. The seizure did not stop with diazepam boluses (to a total of 50 mg), so the patient was re-intubated and a thiopentone infusion used to control seizure activity; mechanical ventilation was continued for a further 24 hours. When the thiopentone infusion was stopped, the patient again appeared to convulse, with clonic movements of his limbs, but he also seemed to be aware. Thiopentone infusion was again used to control seizure activity and his lungs ventilated for a further 48 hours. Again, the patient appeared to convulse, but was evidently aware and responding to speech.

At this stage, the abnormal limb movements were correctly diagnosed as generalised myoclonus secondary to cerebral hypoxia and treatment was started empirically with sodium valproate, with some improvement; the patient was still incapacitated by myoclonus, however, and carbamazepine was added. Over several days, the myoclonus continued and ataxia became apparent, while intellectual recovery was normal. It was noticed that anxiety worsened the myoclonus and diazepam was added to the therapy. The patient was referred to a consultant neurologist who diagnosed the Lance-Adams syndrome, started treatment with clonazepam and stopped other therapy. A rapid improvement in the frequency and severity of the myoclonic jerks occurred and the patient was eventually discharged to the care of his family. He continues to suffer myoclonic jerks which have prevented his return to work as an engineer, although his intellect is unimpaired. He has been treated with increasing doses of clonazepam, to which sodium valproate has been added. He notes that alcohol consumption abolishes the myoclonus.

Patient 2

A 46-year-old man with a long history of asthma, but an otherwise unremarkable medical history, apart from heavy alcohol consumption, was admitted to hospital with an acute exacerbation of asthma secondary to a chest infection. He was initially treated with bronchodilators and antibiotics on a medical ward. However, his condition deteriorated until he had a respiratory, then cardiac, arrest. He was resuscitated and transferred, unconscious, to the intensive care unit where his lungs were mechanically ventilated with difficulty because of profound bronchospasm.

After 18 hours of mechanical ventilation, during which time the patient was treated with bronchodilators, steroids and antibiotics, bronchospasm resolved and weaning from the ventilator commenced. After discontinuing neuromuscular blocking agents and sedatives, the patient developed clonic movements suggestive of a grand mal convulsion, which ceased with the injection of diazepam 5 mg. He was therefore resedated and a CAT scan of the head obtained which showed no abnormality. An EEG performed while the patient was still sedated showed no focal lesion, although there were occasional bursts of activity at 7–11 Hz.

After 24 hours, sedation was again discontinued and once more the patient developed rhythmic contractions of his limbs, but appeared to be aware of, and was distressed by, them. With the previous case in mind, the diagnosis of posthypoxic myoclonus was considered and confirmed by a consultant neurologist. Treatment with clonazepam was started and over the ensuing weeks the patient was weaned from ventilatory support. His myoclonus improved although it did not disappear. He was discharged to the care of his family with some residual disability from intention myoclonus, but with his intellect intact.

Discussion

Posthypoxic generalised myoclonus was initially described by J. W. Lance and R. D. Adams in 1963,⁶ who reported a movement disorder occurring after a period of hypoxia in four patients. The four patients were unconscious immediately after the hypoxic incident for a variable time (2–4 days) during which they had generalised gross jerking movements of the face, trunk and limbs. After recovery of consciousness, the patients continued to experience abnormal clonic muscle movements and it became apparent that myoclonus was initiated by attempted coordinated muscle contraction. Over a period of months to years, the frequency, site and duration of the myoclonus became more restricted. Coughing, yawning, loud noises or events that startled the patient elicited myoclonus, as did any attempt at coordinated movement; the more precise the movement was, the worse the myoclonus. Speech was slurred and slow and the patients were ataxic. Patients could be induced to relax sufficiently to abolish these jerks, as long as they moved slowly. It was particularly striking that they demonstrated no other sign of neurological dysfunction except myoclonus and ataxia; in common with our patients, evidence of intellectual impairment was slight.

The original description of the Lance-Adams syndrome⁶ and later studies of posthypoxic myoclonus^{7,8} suggest that myoclonus originates from afferent impulses from the cerebellum and reticular formation resulting in synchronous and repetitive firing of cells in the thalamus, relayed down the corticospinal tract as myoclonic impulses. Levels of 5-hydroxyindolacetic acid, a metabolite of serotonin (5-hydroxytryptamine) are low in cerebrospinal fluid samples from patients suffering from posthypoxic myoclonus, but were not measured in our patients. Intravenous infusions of serotonin and oral serotonin have been used to abolish muscle jerks successfully,^{7,8} but at the cost of unacceptable side effects of nausea, vomiting and altered mental state. Clonazepam⁸ appears to be the treatment of choice since it is effective, has minimal side effects, and results in increased cerebrospinal fluid levels of 5-hydroxyindolacetic acid, suggesting a beneficial effect on serotonin metabolism or production. Anticonvulsant treatment alone does not appear to have conferred the same benefit on these patients.

The two patients described in this case report have features remarkable in their resemblance to those of the original report of the Lance-Adams syndrome. Both were initially comatose, and were thought to experience generalised convulsions until it became clear that they were conscious during these episodes of myoclonus. Improvement occurred in the abnormal movements with

time and with clonazepam, so that on hospital discharge myoclonus was present only on initiating purposeful movements and only in the muscle groups involved in making the movement. They were both discharged home and neither suffered apparent intellectual impairment.

It is interesting that both of these patients were asthmatics with a precipitating pulmonary rather than cardiac cause of their arrests. Both had a history of heavy alcohol ingestion, but any association with subsequent events is highly speculative.

Improvements in cardiopulmonary resuscitation and improvements in the community provision of emergency medical services, will inevitably lead to intensive care units having to deal with more patients admitted after successful resuscitation. Patients' lungs are frequently mechanically ventilated for 48 hours after arrest to optimise recovery from cerebral hypoxia. In this situation, when the patient is intubated, sedated and artificially ventilated, perhaps with the use of muscle relaxants, it is not easy to distinguish between posthypoxic convulsions and generalised myoclonus. In both our patients, sedation initially masked the fact that although their limbs were twitching they were not unconscious.

Recognition of the Lance-Adams syndrome is fundamental to initiating appropriate treatment early. Failure to recognise the syndrome may result in inappropriate anti-convulsant therapy and delayed treatment, unnecessary prolongation of mechanical ventilation with its inherent risks, and prolongation of distress for the patient's relatives, not least from the possibility of incorrect conclusions about the neurological prognosis.

Acknowledgment

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Potential errors in pulse oximetry

I. Pulse oximeter evaluation*

A. C. RALSTON, R. K. WEBB AND W. B. RUNCIMAN

Summary

There is no absolute reference for oxygen saturation, although multiwavelength in vitro oximeters are accepted as the 'gold standard'. Regardless of whether fractional or functional saturation is used by manufacturers to calibrate their oximeters, evaluation against fractional saturation is recommended since this is the clinically relevant variable. The use of standard notation and comparisons based on bias and precision is recommended. The accuracy of pulse oximetry is intrinsically limited by the use of only two wavelengths, and is dependent on the initial calibration population. The empirical algorithms used to convert the signal to its 'readout value' and the quality control of hardware may both be important sources of variability between oximeters. Change in blood temperature may introduce errors in pulse oximeter and in vitro oximeter saturation readings, but these will be clinically insignificant. Changes in blood pH should not decrease pulse oximetry accuracy.

Key words

*Equipment; pulse oximeter.
Measurement.*

Many recent papers have dealt with the accuracy of pulse oximetry and comparisons of different brands under various conditions, but there are many potential sources of error that need to be considered for the evaluation and appropriate use of pulse oximeters. There is widespread confusion about what pulse oximeters actually measure and which reference oximeter to use. The use of different notations and statistical methods have made comparisons between studies difficult.

This paper is one of a series: two companion papers (which compare and rank, under conditions of poor perfusion, 20 pulse oximeters with finger probes)¹ and 10 with ear, nose or forehead probes (in press). The series aims to review all the important sources of error in pulse oximetry and to discuss their clinical significance. Other papers consider the effects of changes in saturation and signal quality; the effects of dyshaemoglobins, dyes, other pigments and extraneous factors; and present a clinical overview of the significance of all the issues discussed.

Theory of pulse oximetry

Two wavelengths of light are transmitted from light emitting diode (LED) sources and pass through an arterial bed, usually in a finger or earlobe. The attenuated light is received by a detector and converted into an electrical

signal which is analysed by a microprocessor to give pulse rate and oxygen saturation readings. The two wavelengths used are differentially absorbed by oxygenated haemoglobin (HbO_2) and de-oxygenated, or reduced, haemoglobin (Hb). Assuming there are no other major haemoglobin species, the amount that one wavelength is attenuated compared to the other gives the fraction of haemoglobin saturated by oxygen $[\text{HbO}_2]/([\text{HbO}_2] + [\text{Hb}])$, where the square brackets denote concentration.

Both wavelengths are also absorbed by venous blood and tissues. The attenuation of the light is analysed over a full pulse beat to make the saturation measurement independent of these factors. The total absorption of the light has a constant component from the tissue and from steadily flowing venous blood, and a changing component as a result of pulsation of arterial blood. The constant component is subtracted from the total, so that the net absorption of each wavelength can be attributed to arterial blood only. The two LEDs are cycled on and off 480 times per second (for a mains power frequency of 60 Hz) or 400 times per second (for a mains power frequency of 50 Hz), with only one being on at a time.² This enables a single detector to be used to sample first one wavelength and then the other. The detector measures the background level of ambient light after the two LED 'on' periods. This is subtracted from the transmitted LED signals so that

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changing background levels are not included in the signal absorption calculations. This method can fail when the ambient light is almost in phase with the LED cycle, and can lead to erroneous saturation values. The pulse oximeter detector cycles run at a multiple of the local mains power frequency to reduce this interference.

The saturation values that are displayed are not instantaneous but are averages taken over 3 to 10 seconds to help reduce the effect of pressure wave variations due to motion of the subject. To reject fleeting high or low readings, weighting is used by some manufacturers to adjust the contribution of values according to how close they are to the previous average.

These methods are explained in more detail in review articles by Tremper and Barker³ and Wukitsch *et al.*²

Reference oximeters

There must be a direct oxygen saturation measurement for comparison to determine experimentally pulse oximeter accuracy. The 'gold standard' for this is generally accepted to be a multiwavelength *in vitro* oximeter. This instrument transmits light of various wavelengths across a sample of blood. The principle is the same as for pulse oximeters except that the attenuation of the beam from the entire blood sample is measured, in contrast to pulse oximeters which analyse only the changing blood volume within a volume of tissue.

Examples of commercially available oximeters are the Instrumentation Laboratory IL 282 and 482 Co-oximeters (Lexington, MA, USA), and the Radiometer OSM-2 and OSM-3 Hemoximeters (Copenhagen, Denmark). These instruments actually measure slightly different quantities in a blood sample, and there is debate which is more appropriate to use as a reference (see below).

The Radiometer OSM-2 uses two wavelengths of light to determine the percentage of HbO₂ relative to the amount of Hb, and ideally calculates $[\text{HbO}_2]/([\text{HbO}_2] + [\text{Hb}])$. This saturation is known as the functional saturation, but blood, even from 'normal' volunteers, usually contains small amounts of other haemoglobin species, such as carboxyhaemoglobin (HbCO) and methaemoglobin (MetHb), and these affect the absorption of both wavelengths. The saturation measured by the Radiometer is true *functional* saturation only if there is no HbCO or MetHb in the blood sample. If blood is drawn into capillary tubes coated with sodium dithionite to reduce the haemoglobin, the HbCO content can also be measured in a second analysis.

The IL 482 and 282 models⁴ use four wavelengths of light (535.0, 585.2, 594.5, 626.6 nm) to detect the concentrations of HbO₂, HbCO, Hb and MetHb, and give the oxygen saturation as a percentage of the total haemoglobin, i.e. as a percentage of the sum of the four species. This saturation is known as the *fractional* saturation, which is displayed by the Radiometer OSM-3 using six wavelengths. Pulse oximeters are empirically calibrated by the manufacturer against an *in vitro* oximeter. It is therefore important to examine the realistic accuracy limits of *in vitro* oximeters.

The IL 482 operator's manual⁴ claims an accuracy of 1% (95% confidence limits) and precision of 0.5% for HbO₂ measurements for samples with 0–10% MetHb and a haemolysed pH of 7.0–7.4. Outside these MetHb and pH ranges the pH sensitivity of methaemoglobin can cause significant changes in absorption at all four wavelengths.

Accuracy is also compromised by the presence of high lipid levels (which can cause scattering of light), bilirubin, intravenous dyes, sulphaemoglobin and fetal haemoglobin. (The latter four are discussed briefly elsewhere in this series.)

It is not feasible to validate the claim of 1% accuracy, since we know of no quality control sample of accurately known or measured saturation that can be used to check this. Haemoglobin in titrated blood can be fully saturated with oxygen only when there are no other haemoglobin species in the blood, and cannot be easily partially saturated for daily multipoint calibration. Nevertheless, it seems reasonable to accept the accuracy claim, given the high degree of reproducibility of results (within 1%) and the standing of the manufacturers in this area.

Notation and performance criteria

Hundreds of letters and papers are published on the subject of pulse oximetry, and many of them use quite different notation to describe measured and actual quantities. Researchers involved in clinical trials have used a variety of ways to analyse data, which makes comparisons between trials difficult without using the original data. Functional and fractional saturations as measured by a cooximeter and the approximation to functional saturation measured by a two-wavelength Hemoximeter have all been used as the reference quantity. Pulse oximeters are initially calibrated against functional or fractional saturation depending on the manufacturer.

Zijlstra and Oeseburg⁵ recently published a letter on the definition and notation of saturation terms, but unfortunately have a common misconception about what pulse oximeters actually measure. They state that pulse oximeters measure functional saturation, $[\text{HbO}_2]/([\text{HbO}_2] + [\text{Hb}])$, and should only be compared to the functional saturation calculated from the HbO₂ and Hb measurement on a co-oximeter. In fact pulse oximeters only measure a ratio of transmitted red and infrared light intensities, and relate this to a 'look-up' table of empirical oxygen saturation values.² The values in the table depend on the manufacturer's aim of approximating functional or fractional saturation, but will in reality be neither of these unless the dysaemoglobin levels in a subject's arterial blood are exactly the same as the average values of those used for the creation of the 'look-up' table.

The clinician needs to know the fractional saturation, since a high functional saturation can mask life-threatening situations with low fractional saturation (e.g. with high carbon monoxide levels). We have therefore used and recommend the use of fractional saturation as a reference for clinical trials. Pulse oximeters cannot measure this quantity (just as they cannot measure functional saturation), but using fractional saturation as the reference provides the clinician with a realistic assessment of the magnitude of the errors of pathophysiological significance which is likely to be found for the group of patients under consideration.

We have used the symbol Sao_2 to denote functional saturation, as first adopted by physiologists, and $\%\text{HbO}_2$ to represent fractional oxygen saturation. Many groups have adopted a wide variety of symbols which we shall not attempt to catalogue here, except to illustrate this with the use of Sao_2 and FSao_2 by Barker *et al.*⁶ to denote fractional

and functional saturation respectively, compared to the SO_2 and FHbO_2 used by Zijlstra and Oeseburg⁵ to denote functional and fractional respectively.

We agree with Zijlstra and Oeseburg⁵ that the use of the symbol SpO_2 (with the 'p' being for pulse oximetry) is not ideal, since it implies the existence of a new quantity measurable with pulse oximetry, when in fact pulse oximetry aims to approximate functional or fractional saturation. However, the use of the term SpO_2 to denote saturation as indicated by a pulse oximeter is almost universal and we will continue to use it to avoid further confusion.

Comparisons

The presentation of comparative data and subsequent ranking of different brands of pulse oximeters also varies widely. The three basic methods of statistical analysis are regression analysis (correlation coefficient, slope and intercept), the limits of agreement method of Bland and Altman⁷ and the use of bias and precision.

The disadvantages of regression analysis are that high correlation between two variables does not necessarily indicate good agreement even with a slope of near unity and an intercept of near zero, and the results of studies using regression analysis cannot be readily compared to one another.^{3,7}

Bland and Altman devised a method of comparing two indirect methods of clinical measurement⁷ and used pulse oximetry measurements and cooximeter measurements as an example. Some groups have taken up this method, but it ignores the status of the multiwavelength cooximeter as well accepted 'gold standard'. Such cooximeters can measure fractional oxygen saturation with a high degree of reproducibility and avoid many of the inherent problems of *in vivo* measurements with all their uncertainties. Hence we consider cooximetry to be not just another method of indirect measurement of questionable accuracy, but to be close enough to actual saturation for clinical purposes, except in a few specific instances, namely the presence of high levels of fetal haemoglobin, bilirubin and intravenous dyes. Thus this method, however useful in other cases, is not applicable to pulse oximetry assessment when a cooximeter is used as the reference. It may, however, be appropriate when comparing one pulse oximeter to another.

The use of bias and precision is helpful in getting a clear picture of a pulse oximeter's performance and how this compares to other units or other studies. Bias is the average of the differences between the pulse oximeter readings and the cooximeter readings, and precision is the standard deviation of these differences around the average. These quantities may need to be given separately for different ranges of conditions, for example high or low saturation ranges.

A unit may be very precise, so that the results are highly reproducible with a low scatter, but have a high bias so that the results are not centred on the true values. Conversely a unit may be highly accurate, having a low bias, but have poor precision, with values swinging widely from side to side of the true value. Good examples of these cases are given elsewhere.¹ Which of these qualities is more important depends on the clinical application, and ranking of units should rely on subsequent weighting of these. Our

companion paper on pulse oximetry accuracy under conditions of poor perfusion¹ discusses the use of various combinations of criteria in more detail.

Effects of temperature

A pulse oximeter measures the saturation of blood at a temperature in between core and peripheral temperatures, whereas commercially available *in vitro* oximeters measure haemoglobin saturation at 37.0°C. Many factors change simultaneously in the blood, including oxygen saturation when a volume of blood is sealed from air and subjected to a change in temperature.⁸ Thus the saturation measured by the *in vitro* oximeter at 37°C may differ from the actual saturation at the pulse oximeter probe site, especially if the patient is cold as the result of induced hypothermia for surgery, or hyperthermic due to fever.

Siggaard-Andersen *et al.*⁹ state that an oxygen saturation of 90% at 37°C will increase by about 1% when the blood is cooled to room temperature. This effect is even less for lower saturations, with 50% saturation increasing to about 50.5% for the same decrease in temperature. Thus the differences in saturation between blood at 37°C, core body temperature and peripheral temperature are clinically negligible and need not be corrected for.

Siggaard-Andersen *et al.*⁹ also showed that a change in temperature shifts the absorption spectra of HbO_2 and Hb, causing a significant change in HbO_2 extinction coefficient in going from 37°C to 17°C, with a lesser effect on Hb. Values are only given for wavelengths up to 650 nm, so it is not possible to estimate from these data what effect this might have on the accuracy of oxygen saturation measurements made by a pulse oximeter. However, in our intensive care unit¹⁰ and recovery room¹ studies there was no correlation between core temperature, skin temperature or core-skin temperature difference and the differences between SpO_2 readings and IL 482 Co-oximeter % HbO_2 measurements made at 37°C, suggesting that systematic errors from this source are not of clinical significance.

In summary, if a patient is at a temperature other than 37°C this may affect the accuracy of pulse oximetry by shifting the absorption spectra of the haemoglobin species, but clinical data has shown no evidence of this. *In vitro* oximeters have no temperature compensation but the effect of this on *in vitro* measurements is less than 1% error. It is thus reasonable to conclude that overall errors resulting from high or low body temperatures are unlikely to be clinically significant.

Dissociation curves

Oxyhaemoglobin dissociation curves are frequently given in physiology texts and research papers on oximetry. They present a series of curves for varying conditions. For a given PO_2 one can directly read off the graph what the saturation value is for a given temperature, pH, or other variable. However, it is difficult to determine from these graphs what the effect of changing conditions will be on the saturation in a sealed system, such as a blood vessel or syringe. In this case the PO_2 is not kept constant, and will change in response to any change in saturation.

A decrease in temperature for example, from 37°C to 25°C shifts the dissociation curve to the left and raises the oxygen saturation. The increase in oxygen saturation, or

oxygen attached to haemoglobin, means that oxygen is taken out of solution and the PO_2 is lowered. The lower PO_2 opposes the increase in saturation, and an equilibrium point is found on the 25°C dissociation curve. The resultant change in saturation is less than it would be if the PO_2 was fixed.

Using the example given in the previous section on the effects of temperature, blood in a sealed system at body temperature and with a saturation of 50% will increase its saturation to 50.5% when cooled to room temperature.⁹ If the PO_2 of this blood was fixed at about 30 mmHg, for the same decrease in temperature the saturation would increase from 50% to approximately 85%.⁸ Hence these graphs can only indicate the direction but not the magnitude of the effect of changing variables *in vivo* or in sealed blood samples.

Effects of pH

A change in the pH of arterial blood shifts the oxygen dissociation curve and thereby changes %HbO₂,⁸ but the extinction coefficients of the haemoglobin species are not altered and so the accuracy of an SpO_2 reading is not affected.

In vitro oximeters break open the red blood cells by lysis before measuring saturation, and thereby cause a mixing of the intra- and extracellular fluids, the pH values of which differ by 0.2.¹¹ Any change in pH will result in new %HbO₂ and PO_2 values. Thus the %HbO₂ of lysed blood measured *in vitro* is probably slightly different from that of the arterial blood measured by a pulse oximeter *in vivo*. However, pulse oximeters are calibrated against *in vitro* oximeters and so the change in saturation caused by lysing is included in pulse oximeter 'look up' table conversion factors.

Probe reliability

What is quoted as being a single wavelength for an LED, such as the ones used in pulse oximeter probes, is actually the peak wavelength of a narrow range. Ninety-five percent of the LED outputs falls within a range of about ± 30 –40 nm. The absorption coefficients of the haemoglobin species within this range vary somewhat, but since oximeters are calibrated empirically this variation should not matter unless the wavelength spectrum changes after calibration.

The peak wavelength shifts with a change in ambient temperature, increasing by about 0.1 nm for every degree Celsius above 25°C. Thus a change in ambient temperature, which is approximately skin temperature, from 25°C to 35°C would shift the peak wavelength about 1 nm, causing no significant change in absorption coefficients. Severinghaus¹² mentions that age and the increase in LED intensity in response to low light transmission that some pulse oximeters employ can shift the peak wavelength, but does not say by how much.

A much greater variation in peak wavelength of up to 15 nm is caused by differences in manufacture between diodes.¹³ Pulse oximeter manufacturers either reject all diodes outside the acceptable wavelength range or build into the pulse oximeter a device to correct for different wavelength output.³

Reusable probes eventually need replacement, usually

because of damage to wiring. We have seen no confirmed reports of saturation errors from this, except a case of false desaturation when a disposable probe had been re-used several times.^{14,15}

There is a need for portable test equipment that could be used regularly to confirm the integrity of a pulse oximeter and its probe. Munley *et al.*¹⁶ have designed and built such a test object which comprises a hollow dummy finger and a rotating core with a thin slab of pigment. This, surprisingly, causes quite different saturation values for dummy fingers of different pigment concentrations. These authors found a range of SpO_2 readings from 93.5% to 34% for the same core placed in different dummy fingers. The static dummy finger should in theory have no effect on the SpO_2 , which is dependent only on the rotating pigmented core. The SpO_2 , in practice is apparently highly dependent on the pigmentation of the dummy finger, which may be related to the similarly unexpected effects of nail polish. Whether this is a problem of electronics or software, it is a paradox that needs to be addressed by manufacturers if errors due to the light absorption of static pigmentation are to be eliminated.

Pulse oximeters are in general robust enough for frequent transportation and many have self checks of the electronics and software. The largest source of error in probe manufacture is the variation between LEDs, which is determined by the quality control checks by the manufacturer, and may be a point to consider when brands are compared before purchase on the basis of price.

Signal processing

Clinical studies show large differences in the bias and precision of the many brands of pulse oximeter,^{1,3,10} even though they use similar or identical hardware. These differences are almost certainly due to the different algorithms used in processing the measured light intensity signals to obtain a final saturation value. Most algorithms and calibration 'look-up' tables are confidential, so the smaller and newer companies have to start more or less from scratch in their software design. The better models have sophisticated methods which reject noise and motion artefacts, which take frequent running averages of saturation throughout a pulse pressure wave that are carefully weighted to avoid showing sudden changes,² and which take many factors into account to determine when to indicate no reading if the signal quality is too poor. These differences between brands will eventually disappear as companies use only proven optimum algorithms, and the less sophisticated models drop out of the market.

The calibration of pulse oximeters is empirical, and thus relies on the use of a suitable population for building a 'look-up' table that gives the same answers as *in vitro* saturation measurements. Whether functional or fractional saturation is chosen as the reference parameter, the levels of dyshaemoglobins and other red and infrared absorbers in the calibration subjects is crucial to the accuracy of the machine on patients. For example, using healthy, non-smoking volunteers with negligible dyshaemoglobin levels for the calibration means that a pulse oximeter is less accurate when used on day surgery patients who had their last cigarettes over breakfast. It is clearly impossible to match the dyshaemoglobin levels of the calibration and patient populations exactly, which is an intrinsic limitation to the accuracy of pulse oximetry in its present form. The

obvious way to avoid this problem is to use more than two wavelengths in the pulse oximeter probe, and thus enable the accurate detection of more than two light absorbing components in the blood.

Conclusions

Assessment of accuracy is best obtained by comparison of pulse oximetry readings with *in vitro* oximeter measurements, although these are based on the same spectrophotometric principles as pulse oximeters and are prone to some of the same errors. These are discussed in a later paper in this series. Some researchers compare pulse oximetry readings to functional saturation, others to fractional saturation. Some pulse oximeters are empirically calibrated against functional saturation, others against fractional saturation by using volunteers with average dysaemoglobin levels. A pulse oximeter can consistently provide a good measure of either of these only if the blood of the subject has the same dysaemoglobin levels as the average levels of the population group used for creating a calibration 'look-up' table. Fractional saturation is the more useful information for assessing the available oxygen in a patient's arterial blood, and is the quantity we recommend as the reference in clinical trials of pulse oximeters. Ranking of pulse oximeters should be based on the weight put on bias and precision, which may differ for different clinical applications. However, high precision with a moderate bias would seem more desirable than low bias and moderate precision in the context in which pulse oximetry is usually used.

The effect of temperature differences between the pulse oximeter site and the reference *in vitro* oximeter, and the effect of a change in blood temperature on the absorbance spectra of Hb and HbO₂, are likely to be clinically insignificant.

The pH, and hence saturation, of a blood sample may change with the lysis process required for an *in vitro* measurement, but this is taken into account by the initial calibration of a pulse oximeter against these measurements. A change in blood pH does not affect pulse oximeter accuracy.

Pulse oximeter probes are prone to the same wear and tear and variation in manufacturing standards as all monitoring devices. Intrinsic problems stem from the shift in the output spectra of the LEDs with a change in temperature, the inability of the two wavelength system to provide accurate measurements of oxygen saturation in the presence of more than two red or infrared absorbers, and the

mismatch of the dysaemoglobin levels of the calibration and patient populations.

Wavelength reproducibility is an area that will be improved by future technical advances. Similarly, noise and motion artefact rejection algorithms will improve as more complex signal processing techniques can be stored on microchips. The striking differences between the performance of different pulse oximeters will probably decrease as companies adopt the progressively improved signal processing methods, but the problems of absolute calibration in the presence of dysaemoglobins and other pulsatile absorbers can only be achieved by the use of more than two wavelengths.

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Potential errors in pulse oximetry

II. Effects of changes in saturation and signal quality*

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Summary

The published studies of pulse oximeter performance under conditions of normal, high and low saturation, exercise, poor signal quality and cardiac arrhythmia are reviewed. Most pulse oximeters have an absolute mean error of less than 2% at normal saturation and perfusion; two-thirds have a standard deviation (SD) of less than 2%, and the remainder an SD of less than 3%. Some pulse oximeters tend to read 100% with fractional saturations of 97–98%. Pulse oximeters may be suitable hyperoxic alarms for neonates if the alarm limit chosen is directly validated for each device. Pulse oximeters are poorly calibrated at low saturations and are generally less accurate and less precise than at normal saturations; nearly 30% of 244 values reviewed were in error by more than 5% at saturations of less than 80%. Ear, nose and forehead probes respond more rapidly to rapid desaturation than finger probes, but are generally less accurate and less precise. Ear oximetry may be inaccurate during exercise. Low signal quality can result in failure to present a saturation reading, but data given with low signal quality warning messages are generally no less accurate than those without. Cardiac arrhythmias do not decrease accuracy of pulse oximeters so long as saturation readings are steady.

Key words

*Equipment; pulse oximeter.
Measurement.*

The use of pulse oximeters is now widespread, but they cannot, for practical purposes, be calibrated by the user, so it is important that performance be evaluated 'in the field' under both normal (good perfusion, saturation within a normal range and no interfering substances or extraneous factors) and under adverse conditions.

This paper reviews the published studies on the accuracy of pulse oximeters for patients and volunteers under conditions of normal, low and high saturation, exercise, poor signal quality and cardiac arrhythmia. The notation used is that described earlier:¹ functional saturation (SaO_2); fractional saturation ($\%HbO_2$); saturation value indicated by a pulse oximeter (SpO_2).

The large volume of clinical trial data that is published allows manufacturers to re-assess and improve their software frequently. Many of the pulse oximeters used to collect the data referred to in this paper have had software revisions since the original studies were published. Thus the statements of accuracy of any particular unit should not be taken as a definitive measure of its current capability;

rather the data presented should be used as a guide to the potential magnitude of differences in the performance of different pulse oximeters under various adverse conditions.

The majority of the data were available only as scattergrams or calculated linear regression lines and correlation coefficients. Much of the information presented in this paper was derived from these scattergrams and may not be as accurate as the authors' original data. Errors are for this reason grouped into 5% intervals and no attempt is made to determine accuracy or precision from these data.

Normal saturation

'Normal saturation' is defined for the purpose of this review as a fractional saturation of 90 to 97.5%; this corresponds to an arterial oxygen partial pressure of 13.3 to 13.7 kPa, if no other haemoglobin species are present, apart from oxy- and reduced haemoglobin. This may be considered by some to be a rather generous range of 'normal values' but is necessary in part to take into account

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the usual levels of carboxy- and methaemoglobin present in a normal population and in part to allow the inclusion of data from other studies which use saturations of greater than 90% as one of their data groups.

Morris *et al.*² evaluated 15 pulse oximeters on normal healthy volunteers. The absolute mean errors ranged from 0 to 2.3%; the standard deviation (precision) was less than 2% in 10 units, between 2 and 3% in four units and greater than 3% in one unit.

Nickerson *et al.*³ evaluated four pulse oximeters and found that when the saturation was greater than 90% the absolute mean error ranged from 1 to 2.2%; the standard deviation was less than 2% in three units, and between 2 and 3% in the fourth unit. Choe *et al.*⁴ evaluated six pulse oximeters in patients with a P_{aO_2} of greater than 60 mmHg and found the absolute mean error ranged from 0 to 2.7%. The standard deviation was less than 2% for five of the six units.

Re-analysis of data from our previously reported study of pulse oximeter accuracy in intensive care patients⁵ was performed when results were limited to those patients with good perfusion and saturations in the 90 to 97.5% range. The majority of the pulse oximeters had acceptable performance in this range; 10 of the 13 units had an absolute mean error of less than 1.0%; the standard deviation was less than 2% in eight, and between 2 and 3% in the remaining five.

High saturation

'High saturation' is defined for the purpose of this review as a functional saturation of greater than 97%. Pulse oximeters are normally limited by their software so as to not give a saturation reading of greater than 100%; this limits the potential for positive errors and make bias and precision calculations difficult to interpret in this high range. Differences between pulse oximeters are also minimised if the patient population upon which the pulse oximeters are evaluated has a consistently high saturation.

We re-analysed our data in order to determine the tendency of pulse oximeters to read 100% when the saturation is high but less than 100%. All the pulse oximeter finger probe data points with a cooximeter fractional saturation of between 97 and 98% were examined and the number of times that a pulse oximeter simultaneously displayed a reading of 100% was determined. This varied from 13 out of 15 (87%) for one device down to 0 out of 17 (0%) for another, with three of the 20 pulse oximeters reading 100% more than 80% of the time and a further five doing so more than 50% of the time. Five of the pulse oximeters did not read 100% at all when cooximeter saturation was between 97 and 98%. When a similar analysis was performed on the Emergency Care Research Institute (ECRI) data it was found that the same brands of pulse oximeter tended to read 100% in this context (Table 1).

The oxygen dissociation curve flattens out at high saturation levels (>90%) so that a large increase in P_{aO_2} results in a very small increase in saturation. This is of no consequence for most adult patients, but is very important for neonates at risk of retinopathy caused by hyperoxaemia.

One clinical trial used the Ohmeda 3700 and concluded that it was highly accurate and suitable as a detector for hyperoxaemia when the alarm limit was set to 95%.⁷ Another study compared the Ohmeda 3700 and the Nellcor

Table 1. Number of pulse oximeter readings of 100% when cooximeter reading was 97–98%.

Oximeter	Study 1 ⁶	Study 2 ⁵
Criticare CSI 503	0/17 (0%)	–
Engstrom EOS	0/15 (0%)	–
Spectramed Pulsat	0/15 (0%)	0/17 (0%)
Criticare CSI 504	0/14 (0%)	–
Biochem Microspan 3040	0/10 (0%)	–
Radiometer Oximeter	1/17 (6%)	1/11 (9%)
Simed S-100	1/15 (7%)	1/17 (6%)
Invivo 4500	2/15 (13%)	2/9 (22%)
Datex Satlite	3/22 (14%)	1/9 (11%)
Datascope Accusat	3/14 (21%)	4/9 (44%)
Physio-Control 1600	4/16 (25%)	2/17 (12%)
Nonin 8604D	4/16 (25%)	3/9 (33%)
Sensormedics Oxyshuttle	6/16 (38%)	2/7 (29%)
Novamatrix 505	11/22 (50%)	3/17 (18%)
PulseMate Colin BX-5	10/16 (63%)	–
Minolta Pulsox 7	11/17 (65%)	–
Ohmeda Biox 3700	11/15 (73%)	4/9 (44%)
Ohmeda Biox 3740	13/16 (81%)	5/16 (31%)
Nellcor N-200	15/18 (83%)	3/17 (18%)
Kontron 7840	13/15 (87%)	–

N-100 on infants with frequent hyperoxaemic incidents, as determined by transcutaneous oxygen saturation measurements.⁸ It showed that with the alarm limit set to 95% the Nellcor identified all of the 26 hyperoxaemic incidents and gave 25 false alarms; whereas, with the same alarm limit, the Ohmeda identified 13 of 35 hyperoxaemic incidents and gave 29 false alarms. The suitability of the alarm setting for each device should be validated directly.

Low saturation

'Low saturation' is defined for the purposes of this review as a fractional saturation of less than 80%. Pulse oximeters have a high potential for error at low saturations, since ethically manufacturers cannot induce severe hypoxia repeatedly in volunteers for calibration purposes.

Patient data

The accuracy of 13 pulse oximeters at low saturations was determined in intensive care patients.⁵ All units were less accurate and nine out of 13 were less precise than when saturations were greater than 80% (Table 2); eight out of 13 units tended to underread by substantial amounts at low saturations.

Taylor and Whitwam⁹ assessed the performance of five pulse oximeters on patients with saturations down to 63%. There were 16 data points with a saturation of 80% or less. On 10 occasions the pulse oximeters read lower than the cooximeter (–1 to –12%, mean = –6.5%), and on six they read higher (+1 to +18%, mean = 9.8%); five of the six overread data points were with one pulse oximeter. Thus, overall, there was a tendency for pulse oximeters to underread.

West *et al.*¹⁰ recorded S_{pO_2} measured by the Ohmeda Biox III and the Nellcor N-100, and used the Hewlett Packard multiwavelength transmittance oximeter as a reference, for 149 rapid desaturations in patients who had intermittent sleep apnoea. They found that the pulse oximeters tended to underestimate the minimum reached.

Table 2. Accuracy of 13 pulse oximeters using finger probes on patients in the Intensive Care Unit.⁵ Values are expressed as mean (SD).

Brand	Saturation >80% Bias (precision)	Saturation <80% Bias (precision)
Datascope Accusat	-0.3% (1.9)	-7.1% (3.2)
Datex Satlite	+0.0% (2.0)	+1.4% (1.5)
Invivo 4500	-0.3% (1.8)	-0.6% (4.9)
Nellcor N-200	+0.8% (1.7)	-5.5% (3.5)
Nonin 8604D	+1.4% (1.8)	+8.8% (4.8)
Novamatrix 505	+0.7% (1.9)	-8.1% (4.3)
Ohmeda 3700	-1.0% (2.5)	-5.3% (6.2)
Ohmeda 3740	-0.1% (2.8)	-5.5% (1.9)
Physio-Control 1600	+0.0% (1.9)	-6.0% (6.9)
Radiometer Oximeter	-1.5% (1.8)	-6.7% (3.2)
Sensormedics Oxyshuttle	-0.3% (1.8)	-0.4% (1.8)
Simed S-100	+0.1% (2.2)	+1.8% (1.6)
Spectramed Pulsat	+0.7% (1.6)	-3.4% (3.2)

However, these results are presented in the form of regression analysis and are not readily comparable to other studies.

Ridley¹¹ assessed the accuracy of the Nellcor N100E and the Ohmeda Biox 3700 in 25 paediatric surgical patients with cyanotic congenital heart disease. There were 49 data points with a saturation of 80% or less in the evaluation of the Nellcor N100E. The Nellcor overread on 20 occasions (on eight by 5% or less, on seven by 5–10%, and on five by 10–20% (maximum positive error 17%)), and underread on 27 occasions (on 21 by 5% or less, five by 5–10% and on one by 10–20% (maximum negative error -16%)). Similar results were obtained for the 51 low saturation data points with the Ohmeda Biox 3700. The Ohmeda Biox 3700 overread (maximum error 20%) on 18 occasions, and underread (maximum error -11%) on 30 occasions. The underread errors were again on average less than the overread errors (overread errors four by $\leq 5\%$, three by 5–10%, five by 10–15%, six by 15–20%; underread errors 20 by $\leq -5\%$, nine by -5–-10%, one by -10%–-15%). Some of the patients had poor peripheral perfusion, which may have contributed to the pulse oximeter reading errors.

The accuracy of low saturation readings by Nellcor pulse oximeters has been assessed by several groups,^{12–16} all of whom found good correlation (0.91 to 0.999) between the pulse oximeter readings and an *in vitro* oximeter. The combined results show the unit underread on 44 occasions and overread on 52 occasions. Almost 40% of these errors were greater than 5%. This confirms that good correlation may not be an indicator of high accuracy if a significant bias and poor precision also exists. The correlation coefficient for the whole range of saturations in the paper by Boxer¹⁴ was good at 0.95 but the 95% limits of agreement for those saturations below 80% was a poor -9.8 to +7.7%.

Mihm and Halperin¹⁷ monitored 18 patients in respiratory distress with a Nellcor pulse oximeter, and obtained a correlation coefficient of 0.99 between SpO_2 and %HbO₂ measured by an IL 282 cooximeter during three life-threatening arterial desaturation events when the Sao_2 decreased to below 60%. The linear regression line for all 131 data points was $y = 0.97x + 1.51$ with a r value of 0.96. There were nine data points with a saturation of $\leq 80\%$. In four cases the pulse oximeter underread and in five it overread. All errors were less than 5%.

Volunteer and animal data

Some studies have used transiently desaturated volunteers to assess the accuracy of pulse oximeters; these are more relevant to circumstances such as the sudden delivery of hypoxic gas mixtures than to the chronic hypoxaemia of some critically ill patients.

Three groups of researchers used a stepwise reduction in inspired O₂ concentrations in volunteers to assess accuracy of pulse oximeters at each hypoxic plateau. Nickerson³ used four pulse oximeters and found biases (SD) which ranged from -4.9 (2.9)% to +3.0 (2.0)% for saturations of 65–80%. Unfortunately they compared the pulse oximeters with the sum of the oxy-, carboxy- and methaemoglobin concentrations measured by the cooximeter and therefore these findings cannot be compared with the majority of other studies which use %HbO₂ or Sao_2 for comparison. Choe⁴ used six pulse oximeters, and when compared with %HbO₂ had biases (SD) ranging from -0.1 (1.6)% to 1.7 (1.8)% for $\text{Pao}_2 \geq 8.0$ kPa, and from -1.4 (1.6)% to +2.2 (2.3)% for $\text{Pao}_2 < 8.0$ kPa. Correlation coefficients (r) ranged from 0.826 to 0.925 for $\text{Pao}_2 \geq 8.0$ kPa, and ranged from 0.931 to 0.950 for $\text{Pao}_2 < 8.0$ kPa. It appears from these r values that the pulse oximeters more closely followed the cooximeter at lower saturations. However, it is apparent from the scattergrams in this paper that this is not so and is simply a reflection of the statistical fact that one of the factors on which correlation depends is the range of the true quantity in the sample; the larger this range, the higher the correlation coefficient tends to be.¹⁸ The range of true values of saturation for $\text{Pao}_2 > 8$ kPa was approximately 9 (91 to 100)% while the range for $\text{Pao}_2 < 8$ kPa was approximately 22 (69 to 91)%.

Yelderman and New¹⁹ used the Nellcor pulse oximeter and concluded that it accurately and precisely measures Sao_2 down to 70%, but they only present data in the form of regression analysis and do not present any data to support their conclusion about accuracy (mean of differences) or precision (standard deviation of the differences). This again makes direct comparison with other studies impossible.

Another technique is to induce sudden, brief desaturation in volunteers from normal saturation levels. Severinghaus *et al.*²⁰ used this technique to desaturate subjects to 86, 74, 62 and 50%, and assessed the accuracy

of the response of 18 pulse oximeters. Of the 17 units where a correlation coefficient was calculated, 16 were greater than 0.90 and of these 11 were equal to or greater than 0.95. The average bias at a saturation of approximately 55% varied, however, from -21.60 ($r = 0.97$) to 1.86 ($r = 0.98$), the minimum average error being 0.22 ($r = 0.94$). Thirteen of the 18 units had a negative bias. Precision varied from 2.46 to 9.26%. Analysis of this data again demonstrates the problems of using r values to assess accuracy of pulse oximeters as one unit with an r value of 0.97 had calculated limits of agreement of -39.7 to -3.5% .¹⁸

Kagle *et al.*²¹ used a similar technique to evaluate the Ohmeda Biox 3700 (version J finger and ear probe, version XJ1 finger probe) and Nellcor N-100D (finger probe) pulse oximeters and found high correlation (0.98, 0.98, 0.99, 0.99) with *in vitro* saturation measurements. There were a total of 64 data points in the less than 80% saturation range for the three Ohmeda 3700 combinations with the units underreading on 43 occasions and overreading on 30 occasions. The Nellcor N-100D underread on three occasions and overread on 19 occasions.

When the measurable data from the scattergrams in the above papers are pooled there are 244 data points with a cooximeter measured saturation of less than or equal to 80%. The pulse oximeter overread on 126 occasions, underread on 118 occasions and was equal on 10 occasions. The error from these data was 5% or less for 71% of the time, was 5–10% for 24% of the time and was greater than 10%, 5% of the time. There was little difference between the underread and overread groups in regard to the percentages in each error range. The minimum correlation coefficient in all these studies was 0.82 despite the fact that nearly 30% of the values were in error by more than 5%.

It is important not to extrapolate the result of a test which uses only one, or a relatively small number of units, to pulse oximeters in general. The algorithms used are continually changing and vary between brands. This was well shown in the paper by Kagle²¹ where an Ohmeda 3700 with finger probe and Version J software underread on 31 out of 36 occasions, but after modification of its software to version XJ1 it overread on 20 out of 24 occasions.

Sendak *et al.*²² measured the absorption spectra of canine HbO₂ and Hb, to show how close they are to human spectra, and then tested the Nellcor N-100 down to oxygen saturations of 10% in five dogs. They found an average bias (SD) of $+5.5$ (4.2)% for $SaO_2 > 22\%$, and an average bias of $+5.9$ (8.1)% for $SaO_2 < 22\%$.

Glanfield²³ avoided ethical problems and used himself as a subject for prolonged desaturation at high altitude. The Nellcor N-10 was tested in an unpressurised aircraft up to an altitude of 7350 m without supplemental oxygen, and up to 8235 m with 60% oxygen by mask. He concluded that for saturations above 50% the oximeter appeared to be reliable at all altitudes, and that it gave a delayed reading for saturations less than 50%. However, he used no quantitative reference.

Comparison of probe sites

Severinghaus²⁴ found that ear and forehead probes generally had a much faster response to changing SaO_2 values than finger probes. This delay had been found in a previous study²⁵ to be more pronounced in subjects with cold hands (up to 60 seconds), but was reduced after the

introduction of heating pads to the forearms and hands. Kagle *et al.*²¹ found the Ohmeda 3700 finger probe to be on average 24 seconds behind the Ohmeda 3700 ear probe in its response to rapid resaturation. The finger probe was also found to be less accurate during rapid desaturation. West *et al.*¹⁰ found a correlation between heart rate and response delay times. The ear and nasal sensor delays increased slightly with an increase in heart rate, whereas delays with the finger sensor decreased with an increase in heart rate. Delay times ranged from 0.1 (1.6) seconds for the Biox ear probe to 20.0 (5.6) seconds for the Biox finger probe.

West *et al.*¹⁰ also found that measurement accuracy was related to response delay times, with longer delays associated with lower accuracy. The ear probe with the shortest delay had some accuracy problems at low saturations, and the slowest responding finger probe was claimed to be totally inadequate as a monitor of rapid changes in saturation due to its delayed and highly damped response. They found the response time of the Nellcor disposable probes at low heart rates to be better than the reusable probe, which they attributed to better skin contact and alignment of the disposable probe.

Forehead probes have been tested at stable low saturations on volunteers by Cheng *et al.*²⁶ using a commercially available unit and Mendelson *et al.*²⁷ using a custom-built reflectance probe. Both groups found good correlation between the forehead measured values and cooximetry measurements for saturations down to 65%. Severinghaus²⁴ found the accuracy of seven forehead probes comparable to that of finger probes during rapidly induced desaturation in volunteers.

Pulse oximeters are, in summary, poorly calibrated for saturations below 80%. In general, bias is increased and precision is worse than for saturations above 80%, but this is highly variable and depends on the brand. Nearly 30% of 244 values reviewed were in error by more than 5% at saturations of less than 80%. During rapid desaturation ear, forehead and nasal septum probes respond much faster than finger probes, which can have delays of more than 30 seconds. However, forehead and nasal bridge probes may be less accurate than finger and ear probes and should be used with caution (see below). The finger probe response is highly damped during brief transient desaturations and may not show the minimum saturation reached.

Effects of exercise

Ear oximeters are widely used to assess oxygenation in cardiovascular fitness testing, but some doubt has been cast on the validity of ear oximetry during heavy exercise under hypoxic conditions. Powers *et al.*²⁸ and Williams *et al.*²⁹ found that pulse oximeters using ear probes underestimated arterial saturation by 10–15% during heavy exercise. It was suggested that this is caused by reduced ear perfusion in exercise.³⁰ Smyth *et al.*³¹ in contrast found up to 15% overestimation by a pulse oximeter using an ear probe during exercise under hypoxic conditions. It would seem that further studies are required in this area.

Effects of poor signal quality

Poor signal quality occurs when the components of the transmitted red and infrared light beams due to arterial

blood pulsation are small compared to the static components and noise. This may be the result of vasoconstriction, vasodilatation, venous congestion, low blood pressure, high noise levels or other factors. Pulse oximeters may increase the intensity of the outputs of light emitting diodes to obtain a stronger signal, which in turn can affect the accuracy of the emitted spectra.

Patient data

The results of our study on the accuracy of 20 pulse oximeters using finger probes on cold, poorly perfused patients in a cardiothoracic surgery recovery ward⁶ showed a high number of failures to present a reading (i.e. where a pulse oximeter would show a blank display rather than risk presentation of a false saturation value). However, the readings from this study, which included data with poor signal quality warnings, were on average only marginally less accurate than for well-perfused patients in the intensive care unit.

Errors in pulse oximeter readings did not increase significantly with a decrease in patient temperature or blood pressure; this indicates that pulse oximeters generally have an appropriate signal quality threshold below which they show a blank screen, and thus protect the user from possibly erroneous readings as a result of poor perfusion.

Palve and Vuori³² concluded, in a recovery room study of the Nellcor N-100 and Ohmeda Biox 3700 pulse oximeters, that these oximeters were surprisingly accurate on patients with postoperative hypothermia, with accuracies (SD) ranging from 1.8 (2.4)% to 3.9 (1.4)% for finger probes and from 0.9 (2.2)% to 2.1 (1.8)% for ear probes.

Gabrielczyk and Buist³³ similarly found that the Nellcor N-100 was not significantly affected by mild hypothermia. Tremper *et al.*³⁴ investigated the accuracy of the Ohmeda Biox and found that the SpO_2 values were sufficiently accurate for clinical purposes over a wide range of haemodynamic conditions. Paulus³⁵ recommends the use of a paediatric warming blanket wrapped around the forearm as a simple method to increase perfusion to a cold finger if the pulse oximeter signal is weak.

Volunteer data

Some studies using volunteers have investigated the effect of reduced blood flow on the accuracy of SpO_2 .^{2,36,37} However, the use of tourniquets and blood pressure cuffs to occlude blood flow achieves venous occlusion rather than low peripheral perfusion.³⁸

Morris *et al.*² found a marked decrease in accuracy for some units in a study of 15 pulse oximeters, but this was in comparison with a pulse oximeter on the unoccluded arm rather than with *in vitro* blood analysis, and used the rather strict criterion of 2% agreement being acceptable.

Lawson *et al.*³⁷ showed that the Nellcor N-100 gave readings at blood flows of only 4% of initial unoccluded flow, as measured by a laser Doppler flow probe on the thumb. They warn that, even if steady saturation values can be obtained from a pulse oximeter during a critical physiological change or emergency, this cannot be taken as evidence of adequate tissue oxygenation, since flow to the periphery and vital organs may still be very low. Kim and Mathewson³⁹ found that venous occlusion could cause low saturation readings at the base of the finger, and assumed

this to be a result of pulsatile venous flow due to arteriovenous shunting. They suggest this could be a problem if SpO_2 is measured in the hypothenar area in small children.

Comparison of probe sites

If fingers are inaccessible or unsuitable other probe sites may be used, most commonly the ear lobe, nose and forehead (using reflectance instead of transmittance).

Evans and Geddes⁴⁰ used photoplethysmography to compare the vasoactivity of these sites (excluding forehead) and assessed vasoconstriction in response to placing the foot in a bucket of icy water. They found that the nailbed, the finger pad and the nasal septum responded with strong vasoconstriction, whereas the ear lobe was minimally affected. This was, however, only in response to a sudden stimulus and was not the result of hypotension, hypothermia, shock or other stable causes of poor perfusion.

In a study of 20 pulse oximeters under conditions of poor perfusion in cold postoperative patients⁴¹ ear probes ranked significantly lower than finger probes in the number of readings given that were within 3% of the reference and ranked lower for precision ($p = 0.05$). The 'drop out' rates of finger and ear probes were similar. The two nose probes and one forehead reflectance probe often gave grossly erroneous results, and had a higher drop out rate than the finger and ear probes. Palve and Vuori³² also found in a similar study that the nose probe was the least accurate and did not display results any sooner after the conclusion of the operation than finger or ear probes. Finger and ear probes were comparable in both accuracy and drop-out rate.

Hickerson *et al.*⁴² used a disposable finger probe of a Nellcor pulse oximeter on the tongue of a patient with severe burns, and found it gave readings within 1% of arterial blood analysis throughout a 4-hour skin graft operation.

We conclude that finger probes are preferable for the patient with poor perfusion, and that the commercially available forehead and nose probes need further development before they are acceptable clinically, because they can both deliver grossly erroneous values. The use of ear probes did not lower the drop-out rate compared to finger probes, and was associated with less accuracy and precision.

Effects of cardiac arrhythmia

The heart rate derived from a pulse oximeter should match that from an ECG signal for the patient with a healthy heart. If the two differ either of the monitors may be in error because of poor signal quality, or the electrical activity of the heart may appear to be normal while it produces beats with inadequate stroke volume output due to inadequate filling or contraction.

Wong *et al.*⁴³ compared pulse oximetry readings with cooximeter measurements of SAO_2 for 163 patients with cardiac arrhythmias. They found that for the group with a pulse oximeter to ECG pulse rate discrepancy of >3 beats/minute ($n = 24$), SpO_2 measurements were no less accurate than those for the group with pulse rate agreement ($n = 139$), as long as the SpO_2 reading was stable on the pulse oximeter and there was reasonable signal strength.

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Modified occlusion tests for the Bain breathing system

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Summary

Undetected defects in the inner tube of the Bain coaxial anaesthetic breathing system may result in a greatly increased apparatus deadspace. Several authorities have advocated tests intended to detect inner tube problems; however, the efficacy of these tests has never been validated. In this study none of the tests were able to detect all the induced defects. A modification of an existing test using the backbar pressure-relief valve and a new double occlusion test were sufficiently sensitive to detect all defects.

Key words

*Anaesthesia, general; instrumentation.
Anaesthesia, equipment; Coaxial breathing systems.
Safety.*

The Bain anaesthetic breathing system, now widely employed, requires an intact central tube since defects may result in a large apparatus deadspace. Several authors^{1–4} have described tests designed to detect breaks in the integrity of the inner tube, but the sensitivity and specificity of these tests have not been examined; without such information the validity of the tests cannot be assessed.

This study examined the sensitivity and specificity of the Bain system tests, none of which was able to detect all defects. A modification of an existing test,⁵ suitable for all modern anaesthetic machines and capable of detecting all the experimental defects is described. A new and similarly sensitive test suitable for all anaesthetic machines is also described.

Method

Nineteen Bain anaesthetic breathing systems manufactured by Cory Brothers were examined using the four existing tests.^{1–4} A complete Bain system was used to establish the 'normal' outcome for each test. Defective systems were then examined. The defects included an absent inner tube, proximal inner tube disconnection, distal inner tube disconnection, a series of inner tube holes and inner tube breaks.

A total of 12 systems with inner tube holes were examined. Circular holes of 0.75, 2.0, 4.0 and 6.0 mm in diameter were made in the inner-tube at one of three predetermined positions (Fig. 1). The inner-tubes of three further systems were severed at the same positions.

The anaesthetic machine employed was an MIE Cavendish which produces 50 litres per minute fresh gas flow during oxygen flush. A fresh gas flow of 4 litres per minute was used for the inner tube occlusion test³ and the new test employing double occlusion. A 2 litres per minute fresh gas flow was employed for the inner tube occlusion test described by Ghani.⁴

The tests

In this study a positive visual inspection test¹ was recorded when the defect was easily detected by close examination of the 'intact' system.

The oxygen flush test² requires the reservoir bag to hang incompletely collapsed with no fresh gas flow. When the oxygen flush device is operated the reservoir bag collapses, its shoulders are drawn together and move upwards towards the bag mount. The distal end of the inner tube is believed to act as a Venturi, drawing gas from the outer tube and subsequently from the reservoir bag. A positive test was recorded if the reservoir bag filled with gas or did not flatten with apposition of its walls when the oxygen flush was operated. Once this test had been performed with a reservoir bag on the bag mount it was repeated with the bag mount connected to an inclined plane manometer and the subatmospheric pressures generated were measured for each defect.

With a low fresh gas flow, occlusion of the distal end of an intact Bain system inner tube by either a finger or the

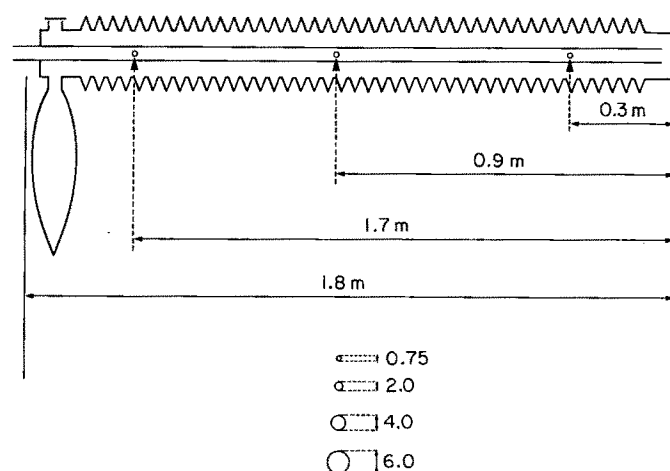


Fig. 1. Defect position and hole size.

plunger of a 2-ml syringe produces a rise of pressure within the inner tube. The pressure gradient across the needle valve is reduced and the bobbin falls in the Rotameter tube. The original description of the test³ states that when the occlusion is released the bobbin rises to the original setting. In our hands, the bobbin overshoots the original setting and the bobbins in the other Rotameters rise briefly from their seatings before they all return to their pre-occlusion positions. We believe that this is due to expansion of compressed gas below the Rotameter bobbins when the pressure is released. A positive test was recorded if the bobbins did not fall when the inner tube was occluded. A record was kept of the operation of the backbar pressure relief valve during inner tube occlusion, a modification of the test which has been described previously.⁵

When the occlusion of an intact inner tube is released a hiss of escaping gas may be noted. The original description of this test used a fresh gas flow of 2 litres per minute⁴ in an attempt to avoid high backbar pressures during occlusion. A positive test was recorded if there was no hiss when the inner tube occlusion was released.

The new test employed in this study was based upon the inner tube occlusion test. However, in addition to occluding the inner tube, the outer tube of the Bain system was also occluded. This double occlusion was achieved in one of two ways. Either the plunger of the 2-ml syringe was cut so that it ended at the level of the outer tube which was then occluded by the operator's thumb, or a cruciate incision was made in a 20-ml syringe plunger rubber which was then fitted to the shaft of the 2-ml syringe plunger. The rubber of the 2-ml syringe was used to occlude the inner tube and the 20-ml rubber occluded the outer tube (Fig. 2). A fresh gas flow of 4 litres/minute was supplied to the inner tube and the expiratory valve was closed. In addition to the changes noted in the previous two tests, the reservoir bag was carefully observed. Any filling of the bag indicates the

presence of a leak between the inner and outer tubes and a positive test result was recorded. In this study Beckton-Dickinson syringes were employed; some minor modification in design may be required if other manufacturers syringes are used.

Results

The results are presented in Table 1.

Visual inspection failed to reveal inner tube holes or centrally placed proximal or distal disconnexions. Absent inner tubes were readily detected and breaks in the inner tube were identified if there was separation of the inner tube at the site of the break.

The oxygen flush test failed to detect holes of 2.0 mm or less, irrespective of their site. Four mm holes at the mid or distal site were undetected. Absence of the inner tube and some proximal inner tube disconnexions could not be detected. Proximally placed 4.0 mm holes, all 6.0 mm holes, circuit breaks and distal disconnection of the inner tube were detected.

The inner tube occlusion tests failed to detect any of the 0.75 mm holes, but reliably detected all other defects.

The failure of the double occlusion test and inner tube occlusion to produce opening of the backbar pressure relief valve reliably detected all the simulated inner tube defects.

Discussion

Coaxial anaesthetic breathing systems provide convenient ways of delivering gases and vapours from the common gas outlet of the anaesthetic machine to the patient. However, this convenience is at the price of greater difficulty in checking the integrity of the system in comparison to conventional breathing systems. Adequate system checks are essential to prevent potentially massive increases in apparatus deadspace and patient morbidity.^{1,5,6}

This study has demonstrated that the tests previously described are insufficiently sensitive to detect all inner tube defects in the Bain anaesthetic breathing system.

Traditional systems may be leak-tested by feel and visual inspection because all parts of the system are readily accessible. This is not the case for coaxial systems. Proximal disconnexions may be more easily detected if the inner tube

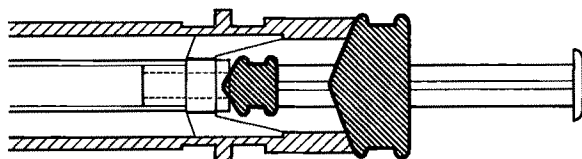


Fig. 2. Double occlusion device.

Table 1. Positive and negative test results for each test performed on complete and defective systems.

Complete System	0.75 mm proximal mid distal	2.0 mm proximal mid distal	4.0 mm proximal mid distal	6.0 mm proximal mid distal	Break proximal mid distal	No inner tube	Proximal disconnection	Distal disconnection
Visual	-	-	-	-	+	+	-	+
O ₂ flush	-	-	-	-	+	+	-	+
Single occlusion	-	+	+	+	+	+	+	+
Single occlusion release	-	+	+	+	+	+	+	+
Double occlusion	-	+	+	+	+	+	+	+
Pressure relief	-	+	+	+	+	+	+	+
Pressure at bag	-11.0	-9.5	-9.5	-9.5	-11.0	-9.5	-1.0	0.3

+ = positive test result
 - = negative test result

mount is proud of the proximal mount for the outer tube so that its full length is visible through the outer tube.

The oxygen flush test failed to detect an absent inner tube; the proximal mount acted as a Venturi, drawing gas from the reservoir bag. Proximal disconnections of the inner tube gave similar results. When the inner tube is disconnected distally the gas flow from the open end is impeded by the mounting for the distal end of the inner tube. This results in a rise in pressure in the outer tube, with consequent inflation of the reservoir bag. Small holes, 2.0 mm or less, are not detected by the test irrespective of site. Holes of 6 mm are detected irrespective of site. As a standard size hole is moved towards the proximal end of the circuit the subatmospheric pressure generated by oxygen flush approaches atmospheric pressure and ultimately may exceed it. This explains why proximally placed 4.0-mm holes were detected by the test whilst those more distally placed were not. We believe this may be a reflection of the resistance of the inner tube distal to the hole. If the tube is long the resistance will be high and gas may flow out of the hole. If the resistance is low gas will flow more readily down the inner tube lumen and create a venturi like effect.

The unmodified inner tube occlusion tests were insufficiently sensitive to detect holes of 0.75 mm in diameter but detected all other defects.

Operation of the backbar pressure relief valve as a result of inner tube occlusion occurred during testing of the complete Bain circuit. The opening pressure was 380–408 cm H₂O (37.3–40.0 kPa) and operation of the valve was clearly audible as a continuous hissing of escaping fresh gas. During testing of all the defective systems the pressure relief valve remained closed. Hence, on machines fitted with a backbar pressure relief valve occluding the inner tube will only result in operations of the valve if the system has none of the defects tested in this study. British Standard BS 4272 Part 3: 1989⁷ requires that all continuous flow anaesthetic machines have a means of limiting common gas outlet pressure to between 35 kPa and 80 kPa. Hence this test is appropriate for all newer machines complying with this standard. There is no reason to believe that damage will result to such machines if this manoeuvre is performed repeatedly. Further reduction in hole size will result in a slower leak through the hole and a higher pressure within the backbar so that very small holes may not be detected.

It has been suggested that in the absence of a backbar pressure relief valve, occlusion of the inner tube is undesirable. Two reasons have been cited. Pressure build up within the Bain system may result in inner tube disconnection.⁸ We believe this is a positive incentive to expose systems to inner tube pressure. If either the proximal or distal connexions are so tenuous that generating a moderate pressure within the system causes a disconnection, they may well come apart and the disconnection be undetected during use. This is a situation analogous to the 'tug test' for pipeline supplies. The second problem is raising backbar pressure to a theoretical maximum of 400 kPa, which may damage both vaporizers and Rotameters. Vaporizers are unlikely to suffer ill effects if they are turned off, or even better, removed from the machine, during the test. Rotameter tubes are unlikely to burst even if transiently exposed to 400 kPa pressure, but may be dislodged from their seals. A rough estimate of the pressure required to produce such damage is

150 to 200 kPa.⁹ Damage may occur to older machines if rubber hoses have been used for internal piping.⁹

A defective Bain system has been detected by one of us (L.F.M.) since completing this study. The system in question had a metal proximal inner tube mount which screwed into the main circuit mount. Examination of the system when it failed the backbar pressure relief valve test revealed that the inner tube mounting had become unscrewed from the main mount. Once this defect was rectified the system performed satisfactorily.

Recommendations

1. All anaesthetic equipment should be tested before use.
2. The integrity of Bain system inner tubes should be tested in one of two ways. If the anaesthetic machine has a backbar pressure relief valve a 4 litre fresh gas flow should be provided to the inner tube and the outlet occluded until the pressure relief valve operates. Failure of the valve to open suggests that there is an inner tube defect.
3. If the machine has no backbar pressure-relief valve the double occlusion test should be employed. The period of occlusion should be kept as short as possible to avoid damage to the Rotameter seals. In our experience a 5

second occlusion of the distal end of a Bain circuit with an initial fresh gas flow of 4 litres a minute failed to produce pressures greater than 80 kPa. At this stage the Rotameter bobbin had fallen to 3 litres per minute.

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Forum

Oxygen concentrators offer cost savings for developing countries

A study based on Papua New Guinea

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Summary

The costs of supplying oxygen to different sizes of hospital in Papua New Guinea have been calculated; the use of oxygen concentrators in this country would reduce the cost of oxygen supplies by between 25% and 75%.

Key words

Oxygen; supplies, concentrators.
Costs.
Developing countries.

Oxygen concentrators provide a possible means of oxygen supply for district hospitals in developing countries.¹ This study compares the cost of supplying oxygen to hospitals of different size using either compressed medical gases (cylinders) or small oxygen concentrators. The price of gases varies in different parts of the world and those quoted in this study are applicable to Papua New Guinea, which was chosen as an example because it has a stable currency, contains the means of production of bulk liquid oxygen, and has an anaesthetic system for its district hospitals based on a regular and reliable supply of compressed oxygen. These features all tend to facilitate the use of compressed medical gases. Although oxygen is produced in the capital as liquid oxygen (the majority for industrial rather than medical purposes), the poor road network necessitates supply of the gas to hospitals in cylinders; it is in any case doubtful whether any district hospitals in developing countries use sufficient oxygen to make supply in liquid form viable even if transportation difficulties were overcome.

Method

Prices of compressed oxygen were obtained from the Papua New Guinea Medical Catalogue² used by all government hospitals in the country. In calculating the cost of oxygen supplies, it was assumed that each hospital would need to carry reserve supplies for 2 months; for the smallest hospitals this might be as little as 3 × 3800 litre cylinders. The cost of transporting cylinders to the hospital was ignored because it is subject to great variation between hospitals (some are supplied by airfreight). Prices have been converted to US dollar equivalents; there is a stable exchange rate 1 Kina (PNG) = \$1.15.

Costs were calculated for the expected oxygen usage of three sizes of hospital, as follows: the smallest hospital (A) uses oxygen for anaesthesia only, at a rate of 3 litres per minute for an average of 3.5 hours per week (2700 litres per month). The source of electrical power for the concentrator is a small (600 W) dedicated gasoline-powered generator.

The intermediate-sized hospital (B) uses oxygen for anaesthesia as above, but for an average of 20 hours per week, and in addition supplies oxygen to a ward patient at 1 litre per minute for an average of 3 days per week (18 720 litres per month). The hospital power supply is a 15 kW diesel generator.

The larger district hospital (C) uses oxygen for anaesthesia as above, but for 40 hours per week, and oxygen to ward patients at 4 litres per minute for 7 days per week (211 200 litres per month). Mains electricity is used to power two oxygen concentrators.

In calculating the cost of oxygen from a concentrator, the following assumptions were made: (1) The concentrator used is of 'domiciliary' size, with an output of up to 4 litres/minute of concentrated oxygen (> 70%), a power consumption of around 350 W, and a general specification and performance similar to that approved by the World Health Organisation for the 'Transoxycon' concentrator.³ The performance specifications of such a machine are aimed at the needs of small hospitals in developing countries. (2) The capital cost of such a concentrator is in the region of \$2400. For the smallest hospitals (hospital A), using oxygen for less than 200 hours per year, the capital cost is divided over 10 years; at the end of this time the machine will have run less than half the hours prescribed for the first service interval. For larger hospitals (B and C) it is assumed that servicing will extend the expected life of the machine to 15 000 running hours and an additional main-

Table 1. Underlying costs.

Cylinder oxygen			
Cylinder size; l	440	3800	7600
Cost of contents; \$	3.80	14.57	26.63
Cylinder rental/month; \$	1.67	2.30	3.39
Oxygen concentrator, purchase price \$2400			
<i>Fuel costs</i>	Gasoline	\$0.56/litre	
	Diesel	\$0.39/litre	
<i>Electricity costs</i>	Mains	\$0.17/kWh	
	Diesel 15 kW	\$0.17/kWh	
	Petrol 0.6 kW	\$0.46/kWh (\$0.28/hour)	

Table 2. Costs related to hospital A.

Oxygen usage per month	2700 litre	
<i>Cylinder supply</i>		
Means of provision	1 × 3800 litre cylinder + 2 spares	
Cost per month	Gas	\$20
	Cylinder rental	\$13.28
Total annual cost	\$399.36	
Unit cost	1.3 cents/litre	
<i>Concentrator supply</i>		
Running hours required per year	182	
Proportion of 10-year capital cost	10% (\$240)	
Cost of electricity from 600-W petrol generator	\$0.28/hour	
Cost of electricity per litre of oxygen	\$0.008	
Annual fuel cost	\$50.1	
Annual cost (capital + fuel)	\$290.1	
Unit cost (litres required)	0.89 cents/litre	
(litres produced)	0.66 cents/litre	

Table 3. Costs related to hospital B.

Oxygen usage per month	18 720 litres	Cost
<i>Cylinder supply</i>		
Means of provision	3 × 7600-litre cylinders 2 × 7600-litre spares	
Cost per month	Gas	\$126.8
	Rental	\$30.5
Total annual cost	\$1889.28	
Unit cost	0.85 cents/litre	
<i>Concentrator supply</i>		
Capital cost concentrator	\$2400	
Running hours required per year	4784	
Proportion of 15 000 hour capital cost	(32%)	\$768
Proportion of 15 000 hour maintenance cost	(32%)	\$192
Cost of electricity (15 kW diesel)	(1674 kWh)	\$285
Annual cost (capital + maintenance + fuel)	\$1245	
Unit cost (litres required)	0.55 cents/litre	
(litres produced)	0.11 cents/litre	

Table 4. Costs related to hospital C.

		Cost
<i>Cylinder supply</i>		
Oxygen usage per month	211 200 litres	
Means of provision	28 × 7600-litre cylinders/month + 28 spares	
Cost per month	Gas	\$747
	Rental	\$177.4
Annual cost	\$11 092	
Unit cost	0.44 cents/litre	
<i>Concentrator supply</i>		
Capital cost of two concentrators	\$4800	
Running hours required per year	10 816	
Proportion of 30 000 hour capital cost (36%)	\$1730	
Proportion of maintenance costs (36%)	\$432	
Cost of mains electricity	\$643	
Annual cost (capital + maintenance + fuel)	\$2805	
Unit cost (litres required)	0.11 cents/litre	

Table 5. Summary.

Hospital	A	B	C
Monthly oxygen usage (litres)	2700	18 720	211 200
<i>Cylinder supply costs</i>			
Annual	\$400	\$1890	\$11 092
Per litre	1.3c	0.85c	0.44c
<i>Concentrator supply costs</i>			
Annual	\$290	\$1245	\$2805
Per litre	0.89–0.66c	0.55–0.11c	0.11c

tenance cost of \$600 is added. (In practice, machines such as the 'Transoxycon' are designed to operate for at least 15 000 hours if serviced regularly). (3) The running costs of the concentrator are taken to be the cost of electricity used; these are calculated according to local fuel costs of different sources of supply, such as mains supply \$0.17/kWh; gasoline \$0.55/litre; diesel \$0.38/litre; running costs for a 600-W portable petrol generator of \$0.46/kWh (\$0.28/hour) and a 15-kW diesel 15-kW generator of \$0.28/kWh. In some cases, use of a concentrator would produce more oxygen than the minimum required; the unit costs are therefore shown both in terms of oxygen required and oxygen produced, the latter being considerably less.

Results

The underlying costs of cylinder and concentrator supply are shown in Table 1. The application of these costs, using the above criteria, to different sizes of hospital, is shown in Tables 2–4 and summarised in Table 5.

Discussion

The figures show significant cost savings in favour of concentrators, ranging from around 25% for the smallest hospitals to 75% for large district hospitals; the potential cash saving in the latter case is more than \$8000 per

annum, a sum sufficient to buy three more concentrators. In many developing countries the savings could be greater, particularly where all medical gases are imported, using scarce foreign currency.⁴ A concentrator-based system has the added advantage of superior logistics in remote locations, since experience has shown that in such circumstances the reliability of oxygen deliveries is poor and supplies often fail; further savings may result if the provision of a reliable oxygen supply removes the need to transport patients to central hospitals for surgery. This study shows that large potential savings may be available, together with increased availability of a reliable oxygen supply. Studies to examine the requirements for setting up a safe and reliable concentrator-based system in the field are now being undertaken by the World Health Organisation.

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Can esmolol manage surgically-induced tachycardia?

Bolus esmolol treatment of intra-operative tachycardia due to surgical stimulation

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Summary

A double-blind, randomised study was conducted to examine the efficacy of a single bolus dose of esmolol in treating surgically-induced tachycardia. Anaesthetic technique was identical in all patients, and consisted of premedication with midazolam and glycopyrronium, induction with thiopentone followed by suxamethonium, tracheal intubation, and maintenance with isoflurane 0.6% (end-tidal) and 60% nitrous oxide in oxygen. Forty-eight patients developed a heart rate of greater than 95 beats/minute or 20% more than pre-induction values at an average time of 34 minutes after tracheal intubation and received placebo (15 patients), esmolol 50 mg (16 patients), or esmolol 100 mg (17 patients). Controlled intervention was instituted if heart rate or blood pressure was not adequate. Both 50 and 100 mg of esmolol resulted in lower heart rates compared to placebo ($p < 0.05$), with no difference between the two esmolol groups ($p > 0.05$). Patients who received placebo had more episodes of medical intervention than those given esmolol ($p < 0.05$). No adverse effects occurred in any patient.

Key words

Heart; tachycardia.

Sympathetic nervous system; β -adrenergic antagonists, esmolol.

Tachycardia and hypertension are undesirable in patients with ischaemic heart disease, but tachycardia increases myocardial oxygen consumption more than hypertension, and results in a greater incidence of myocardial ischaemia.¹ Heart rates (HR) greater than 100 beats/minute have been associated with increased incidences of ischaemia and myocardial infarction.² Intra-operative tachycardia may be caused by severe painful surgical stimulation, unrecognised light anaesthesia, or numerous other causes. Increased anaesthetic depth and adrenergic blockers are frequently chosen to control heart rate. Volatile agents can cause cardiovascular depression,³ while opioids prolong recovery time and cause postoperative respiratory depression.⁴ Most adrenergic blockers outlast the transient stimulus and may cause prolonged hypotension and bradycardia.⁵ Esmolol, an ultrashort-acting cardioselective beta-blocker has an elimination half-life of 9 minutes and plasma concentrations are undetectable after 15 minutes.⁶ The objective of this study was to examine the effectiveness of a single bolus dose of esmolol (50 mg or 100 mg compared to placebo) in treating surgically-induced increases of heart rate regardless of stimulus.

Methods

The study design was double-blind, randomised, parallel and placebo-controlled, and was approved by the institutional Human Studies Subcommittee. Eligible patients were those in ASA classes 2 to 4 scheduled to undergo general or orthopaedic surgical procedures and who had given written

informed consent. Patients with atrioventricular conduction block greater than first degree, atrial fibrillation or flutter, asthma, or severe cardiac, renal, or hepatic disease were not studied, nor were patients in ASA class 1. Premedication consisted of midazolam 0.03 mg/kg and glycopyrronium 0.2 mg intramuscularly 30–90 minutes before induction. Standard monitoring was performed in the operating room (electrocardiogram, Accutor non-invasive blood pressure monitor, SARA mass spectrometer and Nellcor pulse oximeter). Anaesthetic technique was identical in all patients, and consisted of induction with thiopentone 4–6 mg/kg, suxamethonium 1–1.5 mg/kg, tracheal intubation, and maintenance with isoflurane 0.6% $\pm$ 0.06% (end-tidal) and 60% nitrous oxide in oxygen at a flow rate of 5 litres/minute (1 MAC total) for 10 minutes. By definition, we expected 50% of patients to respond by movement in all three groups. The lungs were ventilated to achieve an end-tidal carbon dioxide tension of 4 kPa. Inhalation anaesthesia was continued for a further 10 minutes; if, after this time, systolic blood pressure (SBP) was at least 110 mmHg, patients entered the study if, in response to surgical stimulation, the heart rate (HR) increased either to: greater than 95 beats/minute; or greater than 20% of pre-induction values. Patients did not enter the study if SBP was < 110 mmHg and anaesthesia was lightened. Two qualifying (baseline) HR and SBP values over one minute were obtained and both sets had to meet entrance requirements. The surgical stimulus (incision, retraction, periosteal stimulation, etc.) which caused the HR increase was recorded. Forty-eight out of 152 eligible

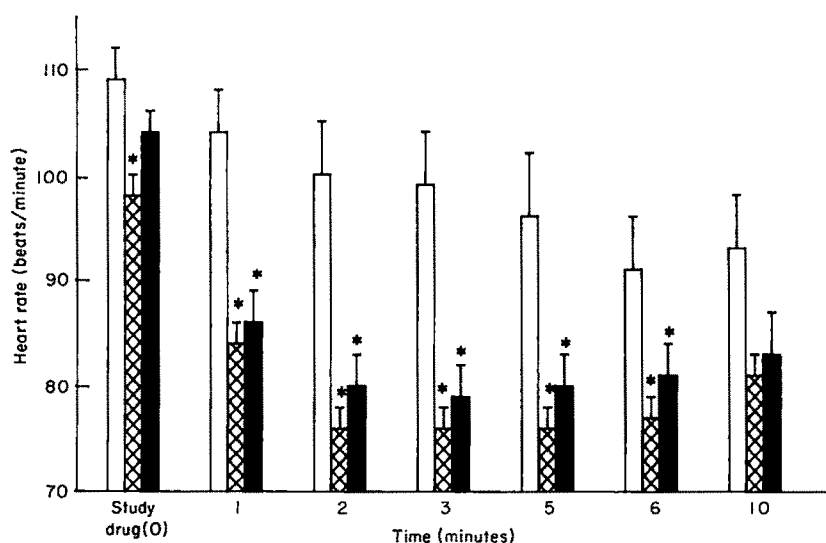


Fig. 1. Heart rate. Both esmolol 50 and 100 mg effectively reduced high heart rates compared with placebo. Bars indicate SEM. □, placebo; ▨, esmolol 50 mg; ■, esmolol 100 mg. *, significant difference between placebo and esmolol ($p < 0.05$).

patients with informed consent met the above criteria and received the same volume of either placebo (15 patients; 10 ml saline 0.9%), esmolol 50 mg (16 patients; 5 ml esmolol 10 mg/ml and 5 ml saline), or esmolol 100 mg (17 patients; 10 ml esmolol) over a 10-second period. The patient was observed carefully for the next 10 minutes, during which time the end-tidal anaesthetic concentration (SARA) was held constant and HR was recorded every 30 seconds and BP every minute. Intervention was instituted as necessary if HR or BP was not adequately controlled by the study drug (HR > 110 beats/minute, SBP > 160 mmHg, ST changes or frequent ventricular extrasystoles); first we deepened anaesthesia, second we gave fentanyl 1.5 µg/kg, and third we gave esmolol 2 mg/kg. Data were analysed using ANOVA and the Newman-Keuls multiple range test. Fisher's exact two-sided test was used to compare the frequency of medical intervention episodes. Kruskal-Wallis

testing was performed to exclude significant differences at baseline. Results were significant if $p < 0.05$.

Results

All three groups (mean value, SD and SEM similar) were comparable in respect of age (49–58 years), height (171–174 cm), weight (74–80 kg), and SBP (127–138 mmHg). Most patients were ASA 2 and distribution of ASA classes was similar in all three groups. Mean (SEM) pre-induction HR was also statistically similar: placebo; 89 (26) beats/minute, esmolol 50 mg; 79 (10) beats/minute, and esmolol 100 mg; 87 (17) beats/minute. Mean HR and SBP are shown in Table 1, and HR in Figure 1. Both 50 and 100 mg of esmolol resulted in lower HR compared to placebo for the 7 minutes after administration ($p < 0.05$). SBP in the esmolol 50 mg group was lower than in the placebo group for minutes 2–10 ($p < 0.05$), with no difference between the 100 mg esmolol group and placebo ($p > 0.05$). Table 2 shows the percentage change in HR and SBP from baseline, and Figure 2 the percentage changes in SBP. The percentage changes in HR in both esmolol groups were different from placebo at minutes 1–6 ($p < 0.05$), and the percentage change in SBP in both esmolol groups was different from placebo at minutes 2–6, and 9–10 ($p < 0.05$). There were no differences between the two esmolol groups in respect of HR or SBP ($p > 0.05$).

The operative stimuli which caused increased HR were surgical incision in 10 patients and postincision pain (dissection, retraction, operative laryngoscopy, bone manipulation or drilling) in 38 patients, and were distributed evenly among groups. Fluid volume administered in kg/hour and volume status were comparable in all groups. Time from tracheal intubation and start of isoflurane to administration of study drug averaged 34 minutes, with a median of 26 minutes and a range of 10 to 130 minutes. Two patients in the placebo group required additional treatment (deeper anaesthesia) during the 10-minute study period; following the end of the 10-minute period an additional eight patients required treatment (deeper anaesthesia) (five in the placebo group, one in the 50 mg esmolol group, and two in the 100 mg esmolol group). Patients given placebo had significantly more medical intervention

Table 1. Mean (SEM) heart rate (HR) in beats/minute and systolic blood pressure (SBP) in mmHg before and after administration of esmolol or placebo at time 0.

Time (minutes)	Placebo (n = 15)		Esmolol 50 mg (n = 16)		Esmolol 100 mg (n = 17)	
	HR	SBP	HR	SBP	HR	SBP
-1	109 (3)	143 (6)	98 (2)	137 (4)	104 (2)	148 (5)
0	109 (3)	144 (6)	98 (2)*	139 (4)	104 (2)	153 (6)
1	104 (4)	148 (5)	84 (2)*	134 (5)	86 (3)*	143 (6)
2	100 (5)	147 (6)	76 (2)*	127 (5)*	80 (3)*	132 (6)
3	99 (5)	146 (6)	76 (2)*	124 (5)*	79 (3)*	132 (6)
4	96 (5)	147 (7)	76 (2)*	123 (5)*	79 (3)*	132 (6)
5	96 (6)	137 (4)	76 (2)*	121 (5)*	80 (3)*	131 (5)
6	91 (5)	139 (5)	77 (2)*	118 (5)*	81 (3)*	132 (5)
7	90 (5)	137 (5)	79 (2)*	120 (5)*	80 (3)*	131 (5)
8	90 (5)	139 (4)	80 (2)*	122 (5)*	80 (4)	129 (6)
9	91 (5)	143 (5)	81 (2)	122 (6)*	82 (4)	132 (6)
10	93 (5)	145 (5)	81 (2)	123 (6)*	83 (4)	129 (6)

*Significant difference between placebo and esmolol ($p < 0.05$).

Minute -1 = baseline = entrance criteria HR/SBP.

Minute 0 = study drug administration.

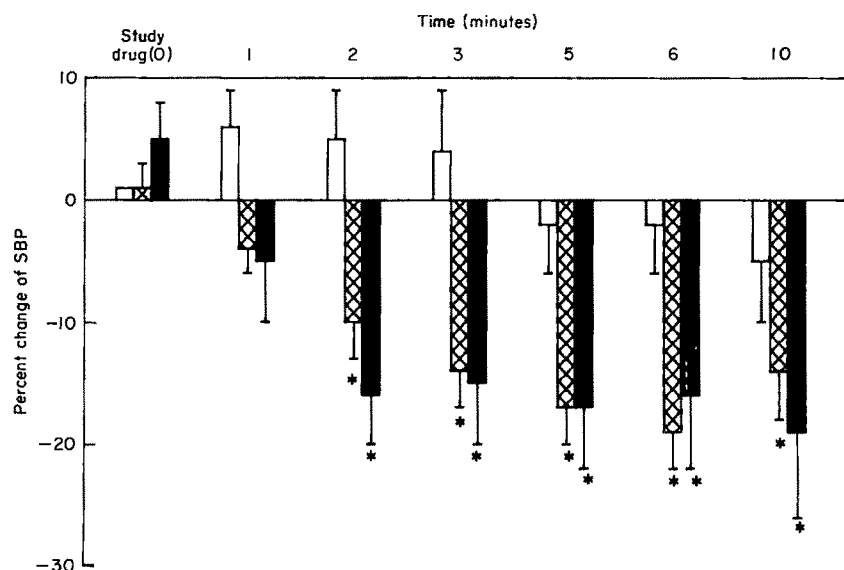


Fig. 2. Percentage change in systolic blood pressure from baseline (entrance SBP). Both esmolol 50 and 100 mg effectively lowered hypertension compared with placebo. Bars indicate SEM. For definition of symbols see Figure 1.

episodes than those in the esmolol groups ($p < 0.05$). No adverse effects occurred in any patient.

Discussion

Both 50 and 100 mg doses of esmolol were safe and effective compared to placebo in controlling increases in HR as a result of surgical stimulation during isoflurane anaesthesia, with no difference between the two doses. We recognise that isoflurane is sometimes associated with fast HR, but all patients received isoflurane. Both doses proved superior to placebo in controlling SBP increases, again with no difference between the two doses, although this was not the primary focus of the study. This suggests that a dose of 50 mg is adequate for controlling tachycardia during surgery.

Esmolol has been used as an infusion to prevent heart rate increases during surgery,⁷ as boluses (50–200 mg) to

prevent the tachycardia and hypertension that accompany tracheal intubation^{8,9} and as a bolus followed by infusion (80 mg + 12 mg/minute) to treat persistent tachycardia.¹⁰ Its use as bolus therapy to treat tachycardia occurring many minutes after tracheal intubation as a result of surgical stimulation has not yet been reported, and was the purpose of this study.

The stimulus that causes increased HR during surgery is frequently transient. This may explain why HR decreased in the placebo as well as in the esmolol group, despite the fact that the anaesthetic level was kept constant. However, the decrease is not always predictable in its timing. A bolus dose of esmolol was usually effective, and is more convenient than setting up an infusion.

Higher doses (range 100 to 200 mg) have been required in the investigations of esmolol^{8,9} to prevent tachycardia in response to tracheal intubation. There may be several reasons for this. First, laryngoscopy and intubation are

Table 2. Mean (SEM) percentage changes in heart rate (HR) and systolic blood pressure (SBP) before and after administration of esmolol or placebo.

Time (minutes)	Placebo (n = 15)		Esmolol 50 mg (n = 16)		Esmolol 100 mg (n = 17)	
	HR	SBP	HR	SBP	HR	SBP
-1	0	0	0	0	0	0
0	0 (0)	1 (2)	-1 (1)	1 (2)	0 (0)	5 (3)
1	-4 (2)	6 (3)	-14 (1)*	-4 (2)	-17 (2)*	-5 (5)
2	-8 (3)	5 (4)	-23 (2)*	-10 (3)*	-23 (2)*	-16 (4)*
3	-9 (3)	4 (5)	-22 (2)*	-14 (3)*	-24 (1)*	-15 (5)*
4	-12 (3)	4 (5)	-22 (1)*	-14 (3)*	-24 (2)*	-16 (6)*
5	-13 (3)	-2 (4)	-22 (2)*	-17 (3)*	-23 (2)*	-17 (5)*
6	-15 (3)	-2 (4)	-22 (2)*	-19 (3)*	-22 (2)*	-16 (6)*
7	-17 (4)	-3 (6)	-19 (2)	-17 (3)	-23 (3)	-17 (6)
8	-17 (4)	-1 (6)	-18 (2)	-16 (3)	-23 (3)	-18 (6)*
9	-16 (4)	-2 (6)	-17 (2)	-15 (4)*	-21 (3)	-16 (7)*
10	-14 (4)	-5 (5)	-16 (2)	-14 (4)*	-20 (4)	-19 (7)*

*Significant difference between placebo and esmolol ($p < 0.05$).

Minute -1 = baseline = entrance criteria HR/SBP.

Minute 0 = study drug administration.

intense stimuli, usually greater than those encountered intra-operatively. Second, in many of the induction studies the patients had only received premedication and thiopentone, and therefore had much less anaesthetic depth than our patients who were already under established anaesthesia of approximately 1 MAC. The greater depth of anaesthesia allowed a smaller dose of esmolol to be effective.

One criticism of our study is that at an anaesthetic depth of only 1 MAC, our patients became tachycardic due to insufficient anaesthesia. However, less than one third of our 152 patients developed a 'treatable' tachycardia, and this tachycardia frequently (38 out of 48 patients) occurred well after incision. The fast HR was distributed equally in all groups. A second criticism is that this was not an outcome study, and therefore we cannot determine whether treatment of tachycardia had any beneficial effect on patient outcome. Third, the small sample population may have limited our ability to detect differences between the efficacy of the two esmolol doses, when in fact a true difference may exist (Type II error). Fourth, isoflurane, which may cause tachycardia, was used. However, patients in all three groups received it.

Coronary artery disease is estimated to be present in 20% of the male population older than 60 years.¹¹ These patients are at risk of ischaemia during surgery,¹² and HR greater than 100 beats/minute may be associated with ischaemia.² It is therefore prudent to treat surgically-induced tachycardia. We conclude that esmolol may be used to treat tachycardia during surgery if clinically indicated, and that because it rapidly treats hypertension, a bolus be considered to gain time for consideration of other therapy.

Acknowledgments

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Premedication: An audit

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Summary

A prospective study of routine premedication management was carried out at a large teaching hospital. Many patients received their premedication at times inappropriate for it to be effective. Several patients receiving regular medication had this suddenly stopped pre-operatively. Prolonged fasting occurred in both elective and emergency groups of patients. Benzodiazepines and opioids were prescribed most frequently and drying agents were used widely. Antacids, H₂-blockers and agents to promote gastric emptying were used very little despite the potential of aspiration in many groups of patients. This audit of activity has revealed several areas of practice that can either be improved immediately or warrant detailed investigation.

Key words

Premedication; opioids, pre-operative care.

Most anaesthetists conclude their pre-operative patient assessment by prescribing 'premedication', the origin of which practice is based in early anaesthetic techniques. Stewart reported to the Scottish Association of Anaesthetists in 1939 that 'no one nowadays will be able to adequately anaesthetise with gas-oxygen alone the unpremedicated abdominal case'.¹ It was necessary at that time to supplement inadequate nitrous oxide/oxygen anaesthesia with opioids and to administer anticholinergic agents, especially when ether was used, to prevent excessive salivation. Today premedication needs to fulfil different goals, which depend on the nature of the surgery and whether it is an elective or emergency procedure. Little has been written about the delivery of routine pre-operative medication despite a plethora of comparative studies that look at the efficacy of various premedication regimens.

The aim of this study was to examine prospectively how patients are managed pre-operatively by members of a Department of Anaesthesia in a teaching hospital. Such an audit could identify problem areas and provide a basis for improvement.

Methods

Approval was obtained from the Departments of Nursing and Anaesthesia and Intensive Care and a simple audit form was developed for use in the admitting area of the operating theatre complex. A one-week pilot study was undertaken to test the data collection forms and familiarise the staff. The ward staff responsible for administration of the premedication were not informed of the survey in order to avoid changes in routine practice. The audit ran for two consecutive months and was intended to include all elective and emergency cases.

The form consisted of 11 questions with a choice of answers that required completion by ticking a series of boxes (Fig. 1). These were completed by operating theatre nurses staffing the patient reception area and the data were analysed using SPSS-X (Statistical Package for the Social Sciences).

Results

Over the course of two months, the total number of cases performed, obtained from operating theatre records, was 1752. A total of 1282 completed questionnaires was

Fig. 1. Premedication audit form.

1. Type of surgery (elective or emergency).
2. Classification of surgery (general, obstetric, orthopaedic, ENT, ophthalmology, vascular, thoracic, neuro, plastic and urology).
3. Age of patient (0–5, 6–15, 16–25, 26–50, 51–70, >70 years).
4. Duration of fasting (<4 hours, 4–8 hours, 9–12 hours, >12 hours).
5. Was operation delayed to fast patient (yes or no).
6. Was patient's usual medication given (yes, no or on no regular medication).
7. Was premedication ordered (yes or no).
8. Mode of administration (oral, intramuscular, intravenous, topical, nebulised, rectal or combination).
9. Drugs given (benzodiazepine—diazepam, lorazepam, oxazepam or midazolam; opioid—pethidine, morphine or papaveretum; antisialagogue—atropine or hyoscine; sedative—trimeprazine, chloral hydrate or phenobarbitone; topical—EMLA cream or GTN patch; antacid/H₂-blocker—cimetidine, ranitidine, sodium citrate or mag. trisilicate; anti-emetic—metoclopramide, prochlorperazine or droperidol).
10. Was premedication given (yes or no).
11. Time since premedication administered (<30 minutes, 30–60 minutes, 61–120 minutes, 120–240 minutes or >240 minutes.)

Table 1. Route of administration of premedicant drugs, and interval between administration and arrival in the operating theatre.

Mode of administration	Time (minutes) from administration of premedication to arrival in theatre				
	< 30	30–60	60–120	120–240	> 240
Oral	19.6% (<i>n</i> = 95)	36.4% (<i>n</i> = 176)	33.7% (<i>n</i> = 163)	8.3% (<i>n</i> = 40)	2% (<i>n</i> = 9)
Intramuscular	17.3% (<i>n</i> = 50)	45.7% (<i>n</i> = 132)	30.8% (<i>n</i> = 89)	4.8% (<i>n</i> = 14)	1.4% (4) (<i>n</i> = 4)
Intravenous	50% (<i>n</i> = 1)	50% (<i>n</i> = 1)	—	—	—
Rectal	—	—	—	—	100% (<i>n</i> = 1)
Topical	—	35.7% (<i>n</i> = 5)	42.9% (<i>n</i> = 6)	21.4% (<i>n</i> = 3)	—
Nebulised	30% (<i>n</i> = 3)	40% (<i>n</i> = 4)	30% (<i>n</i> = 3)	—	—

received, a response rate of 73.3%; this rate was similar in elective and emergency groups, and there was no difference in reporting among the different surgical groups. Elective patients comprised 77.7% and emergency patients 22.3% of the total number. Premedication was prescribed for 67% of all patients who presented for surgery, and elective patients received premedication approximately three times more commonly than those admitted for emergency surgery (75% compared with 25%). Prescribed premedication was not administered to 3.8% of patients before they went to the operating theatre.

Patients who received premedication were administered this via the oral route in 57.9% of cases compared to the parenteral route in 34.6%. The other modes of drug administration were used infrequently compared to the oral and parenteral methods. The topical route was used in 1.7% of prescriptions; the majority of these were for EMLA cream (Astra Pharmaceuticals) (98%) while a glyceryl trinitrate patch was used in the remainder of cases. Rectal administration of medication was used in 0.1% of cases and nebulised administration, for pre-operative salbutamol, used in 1.2% of patients. Combinations of more than one route of administration were used in 4.2% of premedicant prescriptions.

The distribution of the mode of administration differed with age group; 17% of all children less than 15 years of age received a parenteral premedicant compared to 13% who received an oral preparation. In the age group 51–70 years, 17% of adults were given parenteral medication compared to 59.6% premedicated by the oral route. The prescription rates for all age groups were similar and ranged from 57.5% (16–25 years) to 76.4% (51–70 years).

Timing of administration of premedication in relation to surgery is shown in Table 1. A large number of patients received their premedication too late to achieve maximum benefit from its administration.

The majority of patients fasted for between 4 and 12 hours (Table 2). However, one in 20 emergency patients presented for theatre fasted for less than 4 hours, whilst approximately one in 5 fasted for greater than 12 hours.

Opioids were prescribed in 35% of cases (pethidine 43.7%, papaveretum 46.7% and morphine 9.6%) and benzodiazepines in 56.2% (diazepam 95.6%, oxazepam 4% and lorazepam 0.4%). Antiemetic/gastric prokinetic agents were used in 15.0% of elective cases and 6.3% of emergency cases; metoclopramide was used most commonly (98.8%) and prochlorperazine used occasionally (0.2%).

The antisialagogue agents atropine and hyoscine are still in widespread use in all age groups and were part of the premedicant prescription in 15.2% of all cases. Table 3 shows that the majority of opioids are given with another drug, either an antiemetic or a drying agent, whereas the use of these latter agents alone was uncommon.

Antacid prophylaxis was carried out in 51% of obstetric anaesthetic cases, while H₂-receptor antagonists were not used in any branch of elective or emergency anaesthesia. It must be noted, however, that antacid may have been administered subsequently in the anaesthetic room and so was not identified in the audit.

Over one third of patients (38%) were taking some form of regular medication before surgery, but in 45.8% of elective cases and 41.2% of emergency cases was this therapy withheld pre-operatively (Table 4).

Discussion

This audit of current premedication practice highlights several important aspects in the effective administration of pre-operative medication. The aims of premedication vary according to whether the surgery is elective (where it may be more important to lessen anxiety and provide sedation) or emergency (where emphasis is placed on the prevention of vomiting, reduction of gastric volume, increase in gastric pH and provision of analgesia). The patient's regular medication must also be assessed at the pre-operative visit and the decision made whether it is to be continued or stopped pre-operatively.

Oral premedication was the most common mode of delivery, probably due to better patient acceptance and ease of administration by nursing staff, but this delivery system has limitations. White, in a review of premedication

Table 2. Duration of fasting before elective or emergency operations.

Duration of fasting (hours)	Type of surgery	
	Elective	Emergency
<4	0.2% (<i>n</i> = 2)	4.6% (<i>n</i> = 13)
4–8	35.6% (<i>n</i> = 353)	43.2% (<i>n</i> = 123)
9–12	45.8% (<i>n</i> = 454)	28.4% (<i>n</i> = 81)
> 12	18.4% (<i>n</i> = 182)	23.9% (<i>n</i> = 68)

Table 3. Premedication drugs used in combination.

	Metoclopramide	Prochlorperazine	Atropine	Hyoscine	Alone
Pethidine	81.1% (n = 107)	0.7% (n = 1)	6.1% (n = 8)	0	12.1% (n = 16)
Morphine	14.3% (n = 4)	0	46.6% (n = 13)	0	39.3% (n = 11)
Papaveretum	1.4% (n = 2)	0	0	100% (n = 141)	0
Diazepam	10.6% (n = 49)	0	0	0	89.6% (n = 411)
Alone	3	1	1	0	

* Note: two patients received papaveretum, hyoscine and metoclopramide.

practice, stated that most orally prescribed drugs should be given 60–90 minutes before anaesthesia to exert their full effects, whereas intramuscular premedicants needed 30–60 minutes to reach full effect.² One in five of our patients received his (her) oral premedication within 30 minutes of arriving in the operating theatre complex, compared to 17% of the intramuscular group. This may be because of the inappropriate timing of prescriptions from anaesthetic staff, changes in the operating theatre list order or failure of nursing staff to give the medication at the specified time. The most commonly used oral premedications are benzodiazepines, which reach peak plasma concentrations and clinical effects at around the same time when taken by the oral route (time to maximum plasma concentration; diazepam 0.5 hour³ and temazepam 0.3–0.7 hour).⁴ The problem of latency of effect with oral absorption can be alleviated by the use of other routes of delivery without waiting for the development of new compounds, e.g. trans-mucosal temazepam which results in a rapid peak plasma level and clinical effect.⁵ The clinical efficacy of EMLA cream is also dependent on an appropriate application time, yet over one third of applications in this study were given within one hour of arrival in the operating theatre complex, thus reducing its potential efficacy.⁶

Several studies have examined the incidence and potential problems associated with the sudden withdrawal of regular medication.^{7–9} The incidence of concomitant medication and omission rates were similar in these studies to those seen in the present study (38.5% and 44.8% respectively). In many cases the sudden cessation of normal medication may have minimal consequences e.g. vitamin tablets and non-steroidal anti-inflammatory drugs. However, acute withdrawal of cardio-active medications,¹⁰ anti-epileptic agents and psychiatric medications¹¹ can have major clinical consequences.

Opioids are administered to a large number of patients before either elective or emergency surgery. These may be necessary in emergency patients who are in severe pain, but the side effects e.g. nausea, vomiting, pruritus and inhibi-

tion of gastric emptying, need to be considered carefully. In most cases they are administered with an antisialagogue or antiemetic. The use of drying agents has warranted reconsideration with regard to modern anaesthetic techniques; they were prescribed to only 15% of our patients who receive premedication. Atropine can cause a central anticholinergic syndrome of confusion and disorientation in the elderly, an effect which is particularly pronounced with hyoscine. The optimal agent may be glycopyrronium¹² if an antisialagogue is required.

Reduction in the risk of regurgitation is attempted by a combination of pre-operative fasting (usually a period of 4–6 hours) and pharmacological methods. These include drugs that promote gastric motility and alter gastric pH and volume (antacids and H₂-receptor blockers). In this study one in 20 patients who presented for emergency surgery had fasted for less than 4 hours. Gastric prokinetic agents (e.g. metoclopramide and cisapride)¹³ can be used to promote gastric emptying in the presence of a full stomach, yet just over 6% of emergency cases and 15% of elective cases were given this type of medication. These agents are most often given intramuscularly with an opioid which may negate the desired anti-emetic or prokinetic effect. H₂-receptor antagonists that reduce gastric volume and pH have been shown to be of value in areas where the risk of regurgitation is high, e.g. obstetrics.¹⁴ However, during this survey no emergency or elective patient received this form of therapy. This may in part be explained by geographical differences between Australia and British practice in the prophylaxis of acid aspiration.¹⁵

Approximately one in five patients fasted for periods greater than 12 hours. Prolonged fasting has risks; peri-operative dehydration and hypoglycaemia may occur (most likely in paediatric patients and in the elderly) and can lead to nausea, dizziness, malaise, dry mouth and an increase in gastric volume and a decrease in gastric pH. A fasting protocol which allows a light breakfast¹⁶ may be a more appropriate preparation for patients undergoing elective surgery and warrants further investigation.

Table 4. The distribution of concurrent medication in different age groups.

	Age group (years)					
	<5	6–15	16–25	26–50	51–70	>71
Usual medication given						
Yes	100% (n = 3)	57.2% (n = 8)	48.7% (n = 18)	51.9% (n = 50)	61.4% (n = 116)	52.1% (n = 76)
No	—	42.8% (n = 6)	51.3% (n = 19)	48.1% (n = 54)	38.6% (n = 73)	47.9% (n = 70)
% patients receiving concurrent medication	5.9% (3/51)	18.4% (14/76)	20.7% (37/179)	22.4% (104/465)	62% (179/305)	73% (146/202)

It should be noted that, although the response rate to the questionnaire was 73%, the omission rate, derived from the theatre suite register, was similar between emergency and elective groups of patients. This leads us to believe that the results from this study would be applicable to the majority of the patient population.

Increasing awareness of drug costs and limited hospital formularies may dictate the range of premedicant drugs available; these often may be the cheapest rather than the most efficacious. It is important that practising anaesthetists are represented on hospital committees that deal with the ordering of medications and are involved in the review of hospital prescribing patterns. It is also of paramount importance that there is an adequate knowledge among ward medical and nursing staff with regard to the aims of premedication and potential pitfalls in this effective administration.

The value of audit activities was outlined recently by the first report of the Audit Committee of the College of Anaesthetists¹⁷ and several important aspects of premedication practice were highlighted. The majority of patients do receive premedication, but it may be administered at an inappropriate time to have the desired effect. Many patients on regular therapy have this suddenly discontinued pre-operatively, which may have deleterious effects on their peri-operative status. We are currently investigating the specific types of drugs omitted and their peri-operative prescribing pattern. The role of aspiration prophylaxis must be considered, not only in well recognised high risk groups such as obstetric patients but in all emergency cases. The role of antisialagogues and even opioids must be carefully considered as must the role of prolonged fasting in low risk groups.

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Ludwig's angina and tracheal tube obstruction

We would like to report a complication which occurred during fiberoptic intubation of a patient with Ludwig's angina. A previously fit 30-year-old male presented with a 5-day history of toothache and increasing difficulty swallowing and breathing over the preceding 12 hours. Associated with this was extreme swelling of his neck extending to his face and chest. Mouth opening was also severely restricted to about 2 cm. A diagnosis of Ludwig's angina was made by the facial surgeons and an anaesthetic opinion requested. It was decided, after assessment, that urgent tracheal intubation was indicated to secure the patient's airway. Intravenous access was established and the patient taken to the operating theatre.

We decided that an awake fiberoptic intubation was the most appropriate technique. The patient was connected to ECG and pulse oximetry monitoring, his upper airway was

prepared with 10% cocaine solution via the more patent nostril and 10% lignocaine spray to the oropharynx. A transcriothyroid injection with 4% lignocaine was also performed. The trachea was intubated with an Olympus LF-1 4-mm fiberoptic laryngoscope, with difficulty because of severe supraglottic swelling, and a size 7.0 Portex PVC nasotracheal tube was railroaded into position. We were, however, unable to remove the fiberoptic laryngoscope from within the tracheal tube and the patient's breathing became totally obstructed. His oxygen saturation decreased to below 75% and both the fibrescope and tracheal tube were removed. His oxygen saturation improved to over 92% after 100% oxygen by facemask. The procedure was repeated using a size 6.5 PVC tracheal tube. The same problem occurred initially, but with force we were able to remove the fibrescope. As the fibrescope was withdrawn,

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the lumen of the tracheal tube could be seen collapsing in and the patient became obstructed again. The problem did not re-occur when we used a size 6.5-mm armoured tracheal tube (Mallinckrodt). Anaesthesia was able to proceed uneventfully for extraction of carious teeth and incision and drainage of submandibular, submental and paratracheal abscesses. The patient's trachea was extubated 48 hours later and he made a full recovery.

It is well known that PVC tracheal tubes collapse and kink easily at body temperature. When used for nasal fiberoptic intubation the compression caused by nasal structures can hinder withdrawal of fiberoptic, a point made at the 3rd National Fiberoptic Symposium, Guy's Hospital. In the case described, compression of the tracheal

tube and hence inability to withdraw the fiberoptic occurred at a lower laryngeal level causing total airway obstruction and hypoxaemia. We therefore strongly recommend the routine use of armoured tracheal tubes in situations where upper airway obstruction is managed with fiberoptic intubation. They are less likely to be compressed or to concertina, as may occur with nonarmoured tracheal tubes when resistance is encountered, apart from the obvious safety factor in reduced risk of kinking.

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A sticking flutter valve

A 61-year-old woman, ASA 2, was scheduled for a thoracic disectomy. Anaesthesia was induced with thiopentone 5 mg/kg, atracurium 0.5 mg/kg and fentanyl; her trachea was intubated and a peripheral oxygen saturation of 100% was confirmed. She was positioned prone, and her lungs ventilated with 35% oxygen in nitrous oxide, supplemented by 0.5–1.5% isoflurane, using a Nuffield 200 ventilator in a circle system with a fresh soda lime canister. Monitoring consisted of direct arterial blood pressure, electrocardiogram, peripheral oxygen saturation, end-tidal carbon dioxide (with waveform display), inspired oxygen concentration and inspired isoflurane concentration.

Thirty minutes after commencement of surgery, by which time fresh gas flows had been reduced to 2 litres/minute, the trace on the capnograph was seen suddenly to change as shown in the Figure. All other vital signs, including end-tidal carbon dioxide, were unchanged during this episode. The soda lime canister was replaced with no effect on the CO₂ trace and the patient's chest was noticed to be moving, indicating adequate ventilation. Fresh gas flows were increased to 6 litres/minute, again with no effect on the CO₂ waveform. The trace changed between abnormal and normal patterns several times over the next few minutes. It was then noted that the flutter valve on the expiratory limb of the circle system was intermittently sticking in the open position causing significant rebreathing. This resulted in the abnormality seen on the CO₂ waveform. The short duration of rebreathing prevented a rise in the end-tidal carbon dioxide concentration.

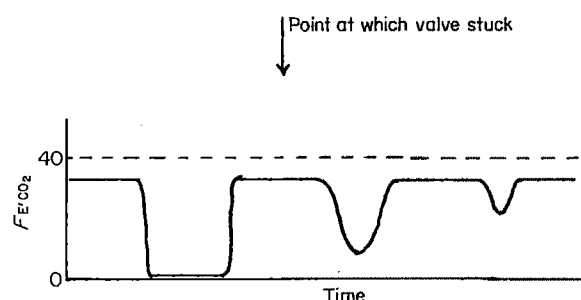


Fig. 1. Diagram of capnograph trace showing development of rebreathing pattern subsequent to sticking of flutter valve.

Circle systems are used increasingly in anaesthesia for reasons of economy and avoidance of pollution. Had a capnograph not been available during this case, it is likely that the faulty valve would have gone unnoticed until the end-tidal carbon dioxide, and hence the arterial carbon dioxide partial pressure, had risen to a dangerously high level. This case illustrates the importance of capnography in situations where low-flow circle systems with soda lime are used.

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Avoidable overinflation of a tourniquet

I would like to draw attention to a pressure gauge, the design of which contributed to accidental overinflation of a pneumatic tourniquet and produced nerve damage. The patient, a healthy, 48-year-old woman had an operation under general anaesthesia on an upper limb. A tourniquet was applied to her arm and inflated after the arm had been prepared for surgery. The tourniquet was attached to a Zimmer gauge and bicycle pump and was inflated by two members of the nursing staff. The first, who was not familiar with the apparatus, asked for assistance from a second more experienced colleague before the task was completed when the needle was seen to be pointing at the 'arm position' on the dial. During the operation, the arterial blood pressure quickly rose to 180–190/100–110 mmHg from a pre-induction baseline of 130/70

mmHg (less marked hypertension is well documented in patients undergoing general anaesthesia with a tourniquet inflated),¹ but decreased promptly to baseline values following a small amount of intravenous fentanyl. After 45 minutes the tourniquet was deflated and the needle was seen to leave the 'arm position' on the dial and rotate anticlockwise past the zero mark and then continue for a further 360 degrees before finally stopping near the zero mark.

On the gauge that was used there is no 'stop' for the needle on the dial face and so it is possible for the needle to point at 250 mmHg ('arm pressure') when it could have made an extra revolution allowing a pressure of 1250 mmHg. This patient suffered a motor and sensory loss distal to the cuff, which has improved since the operation.

It is not clear how fully this will resolve or whether causalgia will appear as a late complication. A case report by Aho *et al.*² describes a similar injury produced when an arm tourniquet was inflated to 500 mmHg instead of 250 mmHg because of a faulty gauge. The surgeon is ultimately responsible for the supervision of equipment he/she wishes to use, but in an operating theatre where tourniquets may not be used frequently, someone may be asked to inflate one who is not familiar with all their potential dangers.

Following this incident we have now decided to stop using the few remaining examples of this type of gauge left in the hospital. This case clearly demonstrates that this style of tourniquet and gauge (which are still used in some hospitals) should be replaced with some of the safer designs currently available. Compensation for an injury of this kind may well exceed the cost of replacing all the old tourniquets in the district with the latest state-of-the-art

failsafe tourniquets. This case also illustrates how a small amount of supervision of the tourniquet (especially those with bicycle pump and a gauge resting on the pillow by the patient's head) by the anaesthetist might help, especially when it is used by operating theatre staff who are not completely familiar with it.

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Sprotte needle for Caesarean section

We read with interest the article by Cesarini *et al.* (*Anaesthesia* 1990; 45: 456-8) about the use of the Sprotte spinal needle for Caesarean section. The authors, however, did not mention the incidence of paraesthesia following spinal needle placement in their patient population, although they reported that the spinal needle was advanced several millimetres into the intrathecal space immediately following identification of cerebrospinal fluid (CSF). We do not agree with this practice because the orifice of the Sprotte needle is more proximal from the distal needle tip than the Whitacre spinal needle; therefore, spinal root trauma could result from advancing the Sprotte needle too far into the subarachnoid space because one cannot guarantee that needle insertion is in the midline. It is possible, while performing subarachnoid block, that the needle may deviate more laterally by advancing it further once it is in the subarachnoid space. Another possibility is that part of the proximal orifice of the Sprotte needle may go out of the subarachnoid space if the needle is more lateral, since the dural sac at the L₃₋₄ interspace is conically shaped. Like Cesarini *et al.*, we have experienced incomplete spinal anaesthesia and hypothesised that the needle's distal orifice was outside the subarachnoid space during local anaesthetic injection. We prefer not to advance the Sprotte needle even a few extra millimetres once CSF flow has been noted because of the potential for nerve root trauma. We disagree with Cesarini *et al.*, that utilisation of the Sprotte needle makes it the 'ideal obstetric technique' for Caesarean section because there is the potential of either advancing the needle too far or not far enough into the subarachnoid space.

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A reply

Thank you for the opportunity of replying to the letter from Dr Ackerman and his colleagues. We agree that 'there is the potential of either advancing the needle too far, or not far enough, into the subarachnoid space,' thus resulting in nerve root trauma or incomplete anaesthesia. This somewhat theoretical statement is not confirmed by the facts and we have never observed any neurological complication with the use of the Sprotte needle for Caesarean section and have never induced paresthesia by advancing the needle 1 or 2 mm into subarachnoid space (700 cases to date). In fact, the characteristics of the Sprotte needle tip makes it the least traumatic of all available spinal needles and this is supported by the absence of postdural puncture headache following its use.

In our practice, the rare occurrence of incomplete spinal anaesthesia seems related to the skill of the anaesthetist, whatever needle or technique is used.

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A 26-gauge pencil-point needle for combined-epidural anaesthesia for Caesarean section

Single-space, combined spinal-epidural (CSE) anaesthesia¹ is becoming increasingly popular, especially for Caesarean section, and because of the high susceptibility to postspinal headache in obstetric patients, the technique has been used to introduce needles as small as 29- or 30-gauge.^{2,3} An alternative solution to the headache problem has been the re-introduction of needles with 'pencil-point' tips first described in 1951.^{4,5}

This pilot study evaluated the use for Caesarean section of 26-gauge pencil-point needles supplied by Portex

Limited in a pack specifically designed for the CSE technique. This pack also contains a standard 8 cm shaft, 16-gauge Portex Tuohy needle, through which the spinal needle protrudes by 12 mm when fully inserted. These 26-gauge pencil-point needles are provided with transparent hubs, which greatly helps the early detection of cerebrospinal fluid (CSF). Note was made of the ease of use of the system; cases in which CSF was obtained at the first insertion of the spinal needle through a correctly placed epidural needle were described as 'technically perfect.'

Table 1. Ease of use of system and incidence of headaches.

Procedure	Number of needles used	Technically perfect (P) or imperfect (I)		Number of headaches	
		P	I	Nonpostural	Postural
Caesarean section	37	37	0	1	0
Caesarean section + tubal ligation	1	1	0	0	0
Total	38	38	0	1	0

Cases which did not conform to this definition were described as 'technically imperfect' and the cause noted (Table 1). Cases in which there was any difficulty in finding the epidural space were not considered to affect the definition, since these are problems of any epidural procedure rather than the CSE technique.

The time to appearance of CSF in the spinal needle hub after removal of the stylet was measured. All patients were followed up at least to the fourth postoperative day and questioned for the presence of headache or neckache. Where these occurred, careful questioning was used to elicit whether they were postural in nature.

All cases fulfilled the 'technically perfect' definition; a correctly placed epidural needle proved remarkably consistent in guiding the spinal needle to the subarachnoid space (Table 1). Only one patient, a sufferer from migraine, had a postoperative headache. She had a typical migrainous headache which was present before the procedure started and continued into the postoperative period. Time to appearance of CSF was short (mean 6.1 seconds, range 4–12 seconds). This is a considerable improvement on the need to aspirate, (or wait 2–4 minutes) to be convinced of the appearance of CSF with metal-hubbed 29-gauge needles. Aspiration of CSF during and after injection to ensure continued correct placement of the needle tip in the subarachnoid space is also easier than with 29-gauge needles.

The pencil-point needle could, in most cases, be felt to pierce the dura mater with a distinct 'click', audible at times. This sensation, disconcerting at first, became reassuring as a clear sign of dural puncture once it became apparent that this was not associated with a high incidence of postspinal headache. Surprisingly, this finding conflicts

with earlier reports of the use of larger pencil-point needles where comment was made on the lack of sensation on piercing the dura;⁶ we cannot explain this difference.

This preliminary study suggests that this combination of needles is a valuable addition to the available CSE equipment. Indeed, as some studies on the use of larger gauge pencil-point needles do report a significant incidence of postspinal headache,⁷ the 26-gauge pencil-point needle may prove to be the ideal size for the combined spinal-epidural technique for Caesarean section.

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Postoperative ventilation and pre-operative anticholinergics

Whilst reading Drs Leigh and Tytler's paper (*Anaesthesia* 1990; **45**: 814–20) I was reminded how useful hindsight is when planning an anaesthetic.

In the 14 cases of morbidity and mortality, which were considered to be wholly, or partially, avoidable, reference is made to the failure of the anaesthetist electively to ventilate postoperatively the patient in seven cases. I would agree that a period of postoperative ventilation is often beneficial, indeed desirable, in the elderly patient with chronic illness, following surgery, particularly emergency abdominal surgery. There will be a large group of such patients during the period of the study who returned to the general ward without problems in recovery, but who would also have benefited from a period of postoperative ventilation on the Intensive Care Unit (ICU) and, indeed, care on the ICU whether their lungs were ventilated or not. However, beds would surely not have been available for all of them? How many of this inevitably large group of patients could have been accommodated? Pressure on ICU

beds may well have been a factor, albeit a wholly unsatisfactory one, in the anaesthetists' decisions to extubate the patients immediately postoperatively in the seven cases referred to.

Unfortunately, if not at the Royal Surrey County Hospital, at the majority of hospitals, there are not enough intensive care or high dependency beds to accommodate all the postoperative patients who would benefit from a stay there. Therefore unplanned admissions from the recovery area will continue; at this stage a bed has to be found. The anaesthetist is, therefore, in an unenviable position, not of his making, and the blame should perhaps be directed elsewhere.

A more specific issue concerns appropriate premedication for a 66-year-old diabetic patient with a history of myocardial infarction for eye surgery. If the authors are suggesting the use of an anticholinergic agent, although they do not specify, pre-operatively, would they not have been concerned about the possibility of inducing a

tachycardia in a patient with myocardial ischaemia? Hindsight would again seem to be a major factor.

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C.F. SWINHOE

A reply

Dr Swinhoe makes a number of points with which we are in sympathy. His response is not dissimilar to those received

after our first article (*Anaesthesia* 1989; 44: 953-8). Nevertheless, a counsel of perfection is still desirable.

The patient for eye surgery had received a sleeping tablet as premedication. However, we find the combination of pethidine and a phenothiazine more desirable and satisfactory in this group of patients and do not use a strongly anticholinergic agent.

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Local anaesthesia for eye surgery

Dr R.A. Fry *et al.* (*Anaesthesia* 1990; 45: 14-17) describe the technique of peri-ocular infiltration of local anaesthetic using Atkinson needles in awake patients for eye surgery. The authors use a large volume (12-20 ml) of an equal volume of 2% lignocaine, and 0.5% bupivacaine and add to the solution 500 IU of hyaluronidase. The authors state that involvement of the anaesthetist in the provision of local anaesthesia for eye surgery has tended to be limited to the interested minority and that the situation had evolved because of the potential catastrophic complication of retrobulbar infiltration especially retrobulbar haemorrhage, optic nerve damage, bulbar perforation, central artery occlusion and intradural injection. They recommend the technique of peri-ocular infiltration which is simple to perform and has a low incidence of even minor complications.

I would like to point out that both peri-ocular and retrobulbar infiltrations are associated with potential catastrophic complications. Both techniques may lead to perforation of the globe, ocular haemorrhage and central artery occlusion. There is the potential complication, with both techniques, of intravascular injection that may lead to immediate convulsions and cardiorespiratory depression. More than one injection may be needed using peri-ocular infiltration that may increase the possibility of intravascular injection.

It is true that retrobulbar and not peri-ocular infiltration is associated with two possible catastrophic complications: intraneural and subdural injection of local anaesthetic which could lead to brainstem anaesthesia. These can happen particularly in an awake patient who is instructed to look upwards and nasally, an eye position that stretches the optic nerve and increases the possibility of intraneural and subdural injections.

In our practice, we administer a brief general anaesthetic using 1-5 µg/kg of alfentanil and a sleep dose of methohexitone intravenously for placing the retrobulbar

block. Under general anaesthesia the eye is in the neutral position, i.e. the optic nerve is not stretched and this decreases the chances of intraneural and subdural injection. Patients do not feel pain associated with placing blocks. They are awake in a short time with a clear mentality and are sedated and cooperative. The main advantages of using retrobulbar infiltration is its reliability, speed of action and that the block is accomplished most of the time by one injection.

Like the authors we prefer to use Atkinson needles. They are blunt, short-bevelled, 1.25-inch long, 23-gauge needles. It is safer to use Atkinson needles compared to sharp needles since they help to identify the different structures of the orbit, but their use in an awake patient is painful. We mix 10 ml of 0.75% bupivacaine plus 150 USP units of hyaluronidase and use 6-7 ml of the mixture for retrobulbar infiltration; 3 ml is used for fascial nerve block on withdrawing the needle. The use of bupivacaine not only provides adequate anaesthesia for long procedures but also prolonged postoperative analgesia and paresis of the orbicularis muscle. This prevents its squeezing action on the globe and may have surgical advantages.

We believe that anaesthetists should be involved in the care of ophthalmic patients, and administer the blocks themselves; they are the most qualified physicians to take care of the associated potential systemic complications of the blocks. We practice in a teaching hospital; this involves teaching new residents every month. We have performed over 700 eye surgery cases, with one retrobulbar haemorrhage, which was not severe and the surgeon was able to perform the scheduled cataract surgery without delaying the case and with no surgical complication.

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Heparin dose response: *in vitro* versus *in vivo* technique

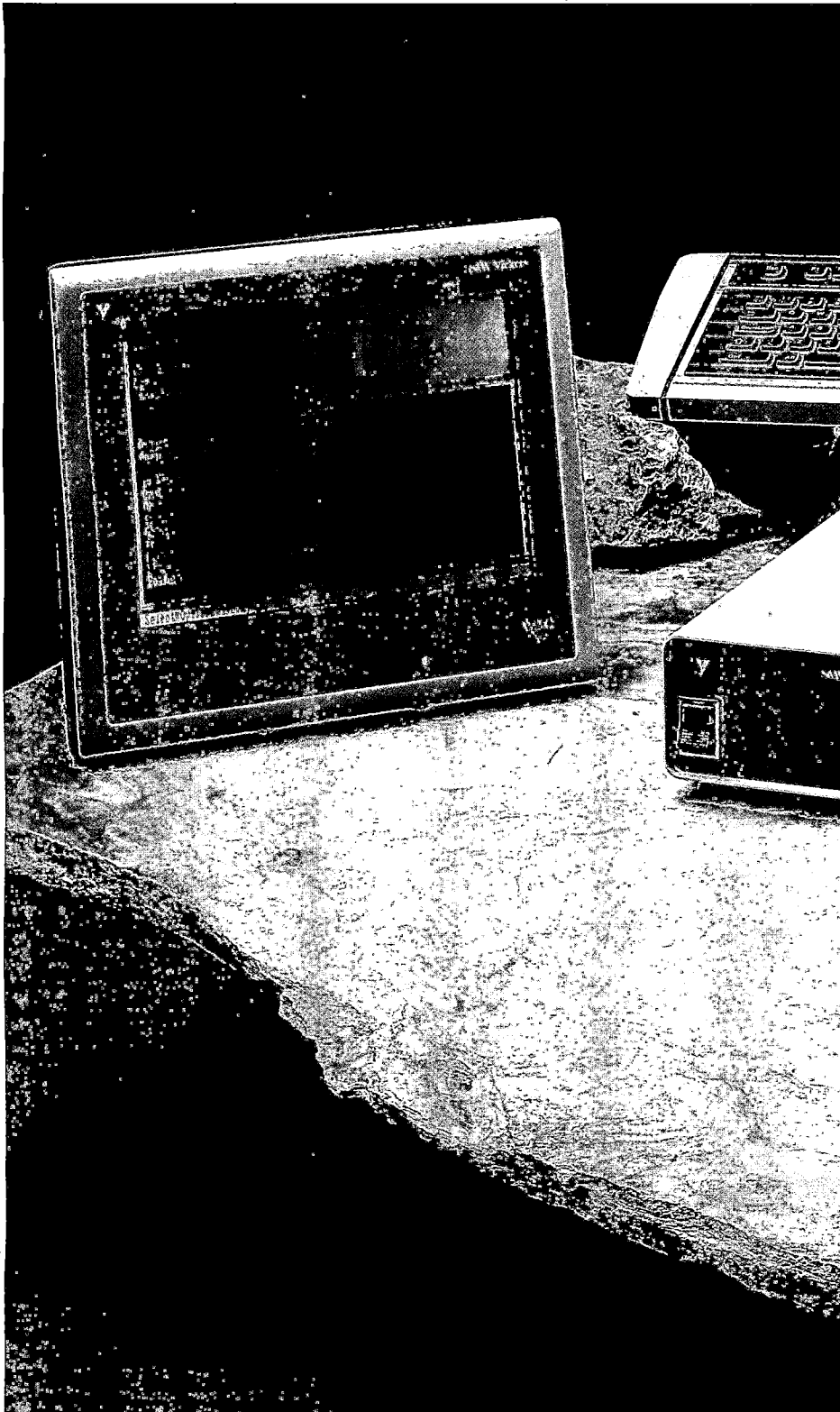
Before introduction of the Hepcon four machine* to our centre, we had been following the method described by Bull *et al.*¹ for calculating the heparin dose response and further management of anticoagulation during cardiopulmonary bypass (CPB). When we started using the Hepcon system we observed that the machine was showing heparin maintenance levels lower than expected. After giving the calculated dose of heparin to the patients, the activated

clotting time (ACT) was consistently lower than 480 seconds. Therefore we conducted a prospective study to compare the heparin dose response by Bull *et al.*'s *in vivo* method with that derived from the Hepcon system (*in vitro*).

Fifty patients undergoing routine coronary artery bypass grafting (CABG) were studied. All the precautions as mentioned by the manufacturer's instruction manual in conducting the tests were taken. The baseline ACT was 113.2 (SD 15.12) seconds by Hepcon cartridges and 119.35

* Hemotec, Inc. Englewood Co., USA.

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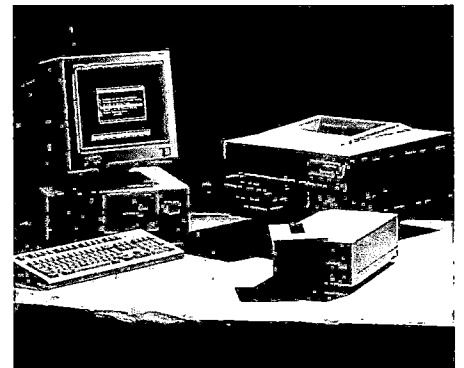


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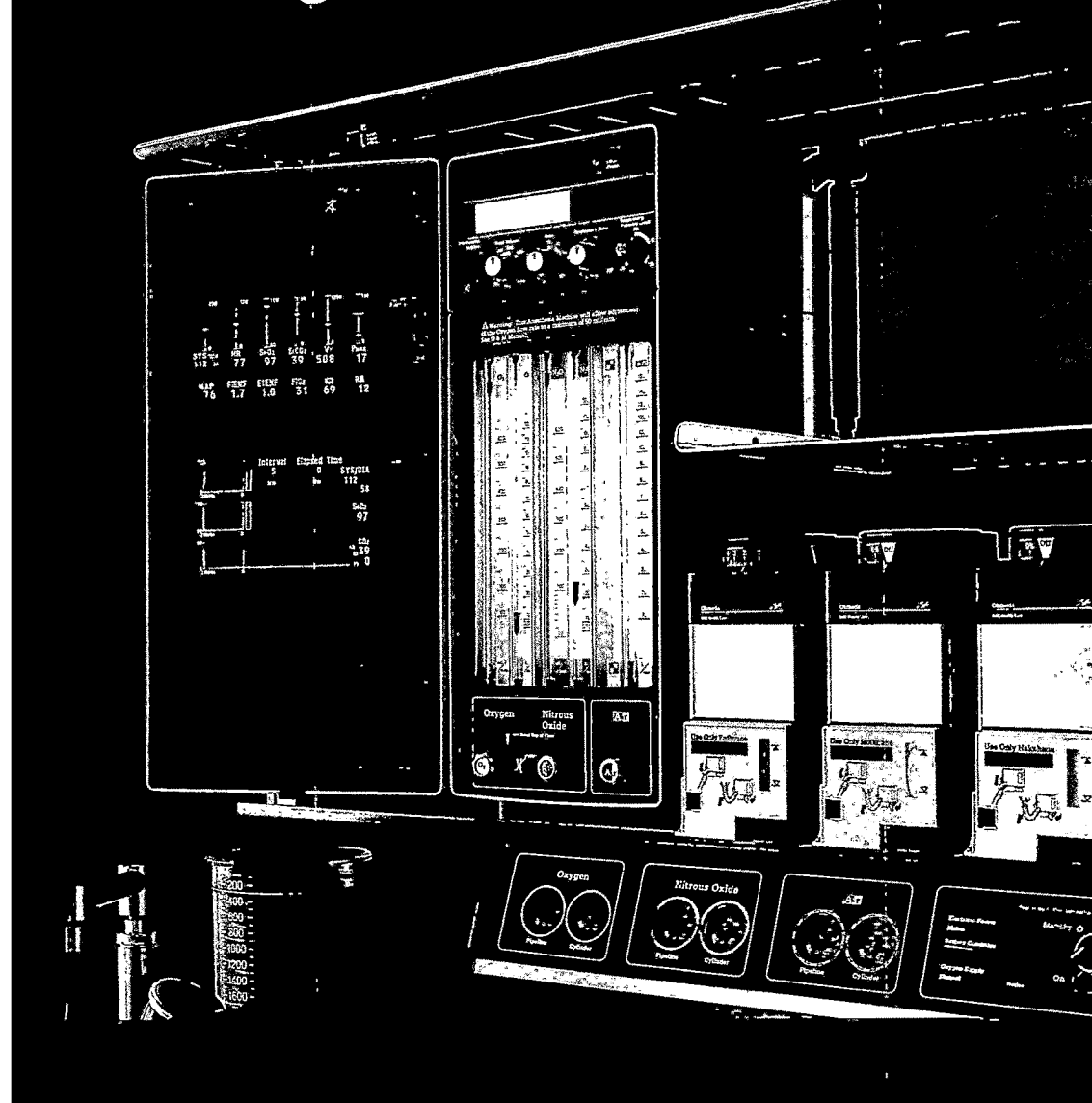
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(SD 14.12) seconds by the Hemotec ACT machine* (not significant). The dose response test performed by the Hepcon calculated heparin maintenance levels of 333 (SD 65) IU/kg. The same dose was given to the patients at the appropriate time and after waiting for 5 minutes. The repeat ACT was 432.75 (SD 72.60) seconds. This was taken as the second point in drawing the dose response curve according to Bull's method (this was slightly modified as the authors mention 200 IU/kg as the initial dose of heparin in their original study). Extra heparin required was 85 (SD 71) IU/kg, which was given to the patients. Repeat ACT after 5 minutes was 510 (SD 34.93) seconds. By Bull's method the patients' heparin maintenance requirements were 394 (SD 67) IU/kg, which was highly significant ($p < 0.001$) when compared to the maintenance levels shown by the machine.

The dose response produced by the Hepcon tells us whether a particular patient is resistant or sensitive to heparin before he/she is subjected to the loading dose. There can be a difference in the potencies of different brands of heparin (one which is being used in the cartridge for the test and the second which the patient is receiving). This is especially so when they come from different sources or manufacturers. The cartridges of the Hepcon system contain USP beef lung heparin. The USP heparin exhibits a potency between 90 and 110% and is prepared, assayed and dispensed in terms of units/ml only.² The heparin IP we have been using is derived from beef intestinal mucosa and potency is determined by biological assay using a reference standard based upon units of heparin activity/mg.

Although the source of both the brands was beef, the organ of extraction was different. Additionally, the potency is known to vary due to quality of bottles and lot number in the same batch.³ Lastly there can be loss of potency if the storage conditions are less than ideal.

It would seem prudent if some method is evolved by which the *in vitro* heparin dose response performed in the operating theatre uses the same brand of heparin which is to be given to the patient. If the brands of heparin used are different, there is bound to be some confusion. We must emphasise that one should recheck ACT before going on to bypass rather than be influenced by 'hitech' machinery.

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Ball-valve effect of a contaminated spinal needle

We wish to report unusual difficulty in using a spinal needle. An 80-year-old male patient was scheduled for transurethral resection. He was obese, confined to a wheelchair because of non-union of a fractured right hip and was also wheezy at rest due to asthma since childhood. He was taking no medication and consented to a spinal anaesthetic.

An SHO anaesthetist made two unsuccessful attempts at dural puncture at the L₂₋₃ interspace in the sitting position with a 26-gauge clear hub spinal needle. Approximately 5 minutes later a second more experienced anaesthetist was summoned who attempted dural puncture at the same site and in the same posture. A small amount of blood was present in the translucent hub, but because the obturator passed freely down the lumen, the same 26-gauge spinal needle was used. As the spinal needle was advanced, a click was felt due to penetration of the dura and the obturator was removed. Free back-flow of cerebrospinal fluid (CSF) occurred. A 5-ml syringe containing 3 ml 0.5% heavy bupivacaine was attached to the hub and 0.2-0.3 ml of CSF was aspirated. However, it was impossible to inject the contents of the syringe through the spinal needle despite considerable pressure. The syringe was disconnected from the hub and its contents were discharged easily into a galley pot, confirming the syringe was functioning normally. The obturator was re-inserted down the spinal needle lumen

easily and CSF flowed back again when it was removed. However, when the same syringe containing another 3 ml 0.5% heavy bupivacaine was re-attached, the plunger could not be depressed at all. The syringe was removed again and very close inspection of the transparent hub of the spinal needle showed that a small (approximately 0.1 mm × 0.2 mm) blood clot was adhering to the wall of the hub alongside the lumen of the needle and was acting as a ball-valve which only allowed flow outwards. This clot was removed by dislodging it with the obturator and then aspirating it up into a new 2-ml syringe along with 1.5-2 ml of CSF. Then the local anaesthetic solution was injected freely and the spinal needle removed. There was no kink in the needle. Spinal anaesthesia proceeded uneventfully. We should point out that the transparent hub of the spinal needle enabled very close inspection of the hub for CSF or blood and so we were able to identify the cause of this problem. This would have been more difficult with a metal-hubbed needle.

We suggest that readers should bear this problem in mind if a bloody tap occurs and there is a delay in re-using the needle. Flushing the needle with saline would have prevented the problem.

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Prolonged local analgesia is not always ideal

Drs Hutchison and Barthram (*Anaesthesia* 1990; 45: 987) reported a case of unilateral spinal anaesthesia in a patient requiring varicose vein surgery in both legs. Under general

anaesthesia, a femoral nerve block was performed on the unanaesthetised side using 15 ml 0.5% bupivacaine. Twenty-four hours later the block had not worn off and

this interfered with mobilisation. The duration of local anaesthesia is influenced by the pharmacology of the agent (protein binding and potency), mass administered, presence or absence of a vasoconstrictor and the site of injection. Peripheral nerve blocks using agents of high potency and long duration, such as 0.5% bupivacaine, are well recognised to last in excess of 10 hours and reports of much longer duration are not uncommon.¹⁻³

Regional anaesthesia has much to offer, both for operative anaesthesia and postoperative analgesia. However, requirements for postoperative mobilisation, especially where there is a need for early weight-bearing should influence the anaesthetist's choice of local anaesthetic and site of injection. This case illustrates well that, as with any form of anaesthesia or analgesia, a longer duration is not always ideal. It is often more appropriate to

block centrally (spinal or epidural) or select an agent of moderate potency and duration, for example lignocaine or prilocaine. Where the block is used to supplement general anaesthesia and to provide postoperative analgesia, using a smaller mass (lower concentration) is another option.

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Laryngeal mask airway for coronary artery bypass grafting

We believe this to be the first report describing the use of the laryngeal mask airway (LMA) for anaesthesia for coronary artery bypass grafting.

The patient, aged 67 years, had an uneventful anaesthetic history. On examination there was nothing to indicate possible difficult tracheal intubation. Pre-operative medication of papaveratum 20 mg and hyoscine 0.4 mg was given. In the operating theatre, intra-arterial blood pressure, electrocardiographic and pulse oximetry monitoring were commenced and anaesthesia was induced with fentanyl 1 mg and thiopentone 125 mg while pancuronium 10 mg was given for muscle relaxation. At laryngoscopy, only the tip of the epiglottis was visible and tracheal intubation proved impossible despite the attempted blind passage of a gum elastic bougie and lighted stylette. Anaesthesia was maintained during these attempts with nitrous oxide, oxygen, and halothane. A LMA size 4 was then easily placed, a secure airway and an airtight seal obtained, and mechanical ventilation of the lungs was commenced. It was not possible to pass a gum elastic bougie through the LMA into the trachea and further instrumentation was rejected to avoid further trauma to the airway since the patient was to receive heparin. Similarly, the possibility that conventional tracheal intubation could be attained during the period of cardiopulmonary bypass when gaseous exchange was independent of the airway was rejected. During the procedure, expired minute volume, expired tidal volume, peak inflation pressures, and oxygen saturation were continuously monitored and arterial blood gas analysis was performed at 15-minute intervals and remained within the normal range.

The LMA has several limitations because of the unreliability of achieving a gastight seal between the respiratory and alimentary tracts. However, there was no gas leak during the operation, which lasted 1 hour 52 minutes, with a cardiopulmonary bypass time of 34 minutes. Peak inflation pressures reached a maximum of 20 cmH₂O 20 minutes after weaning from cardiopulmonary bypass. No evidence was found of regurgitation or inhalation of stomach contents in this patient who had been starved overnight. At the completion of surgery neuromuscular blockage was reversed with neostigmine 2.5 mg and glycopyrronium 0.5 mg. The patient was transferred to the cardiac recovery ward where spontaneous ventilation was re-established. The LMA was removed 20 minutes later. The *Paco*₂ increased to 7.8 kPa after 10 minutes spontaneous ventilation but rapidly fell back to within normal values 5.97 kPa at the next sampling. The patient made a full recovery and the difficulties of intubation which had been experienced were explained to him before his discharge home.

Although we would not recommend the routine use of the LMA for cardiac surgery, if intubation proves to be very difficult or impossible the LMA should be considered as an alternative, but with the understanding that it may not isolate the respiratory from the alimentary tracts as effectively as a properly placed cuffed tracheal tube.

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Oxford OX3 9DU

A. WHITE
M. SINCLAIR
R. PILLAI

Gum elastic bougie and difficult tracheal intubation

I am glad to see that Drs Dogra, Falconer and Latto (*Anaesthesia* 1990; **45**: 774-6 and 977-8) are giving further publicity to the use of the gum elastic bougie for difficult tracheal intubations and stressing the benefit of the quarter-turn anticlockwise twist when railroading the tube along the bougie. When describing this technique in a letter to another journal,¹ my suggestion that one should practice this initially on easy cases was omitted. Now that the success of the -90° rotation has been 'quantified' the technique is presumably respectable enough to be used as a routine when instructing juniors on the management of difficult cases. The -90° rotation proved to be helpful in guided blind intubation in one reported case.² I use the

bougie routinely as an aid to nasal intubation, but the advantage of the -90° rotation in this procedure has yet to be confirmed.

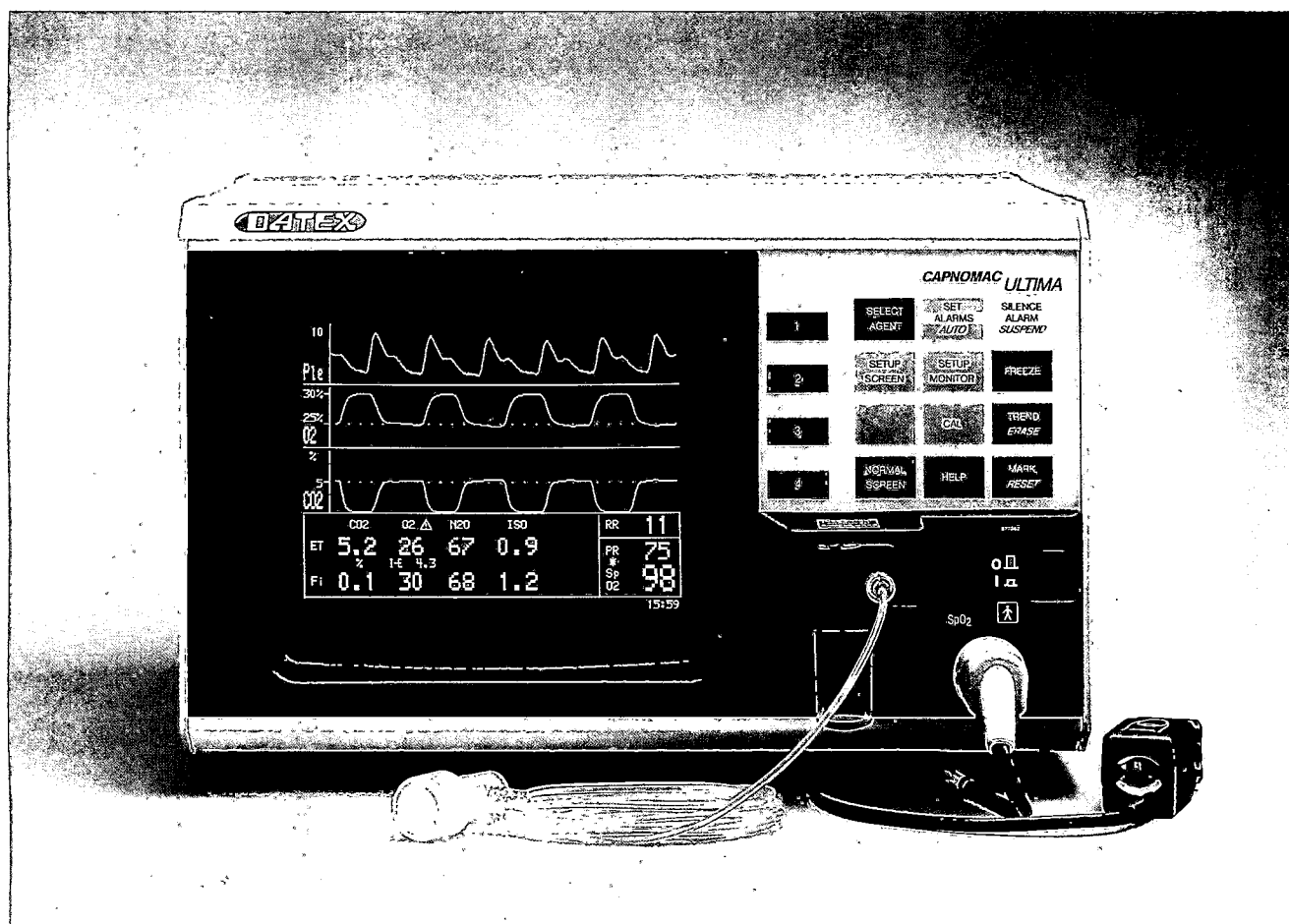
Leicester Royal Infirmary,
Leicester LE2 3JJ

P.S. COSSHAM

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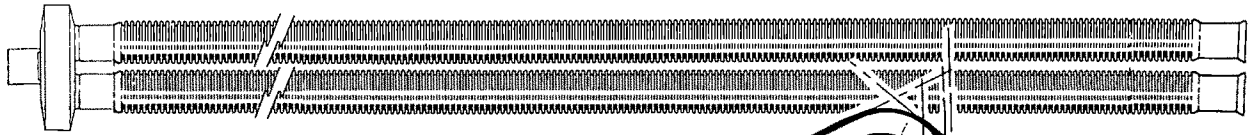
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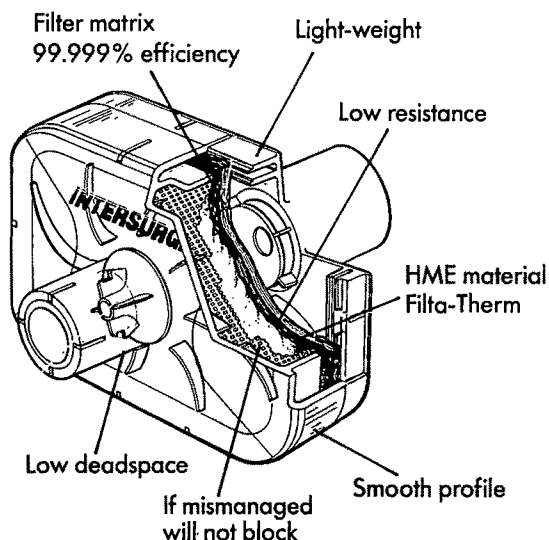
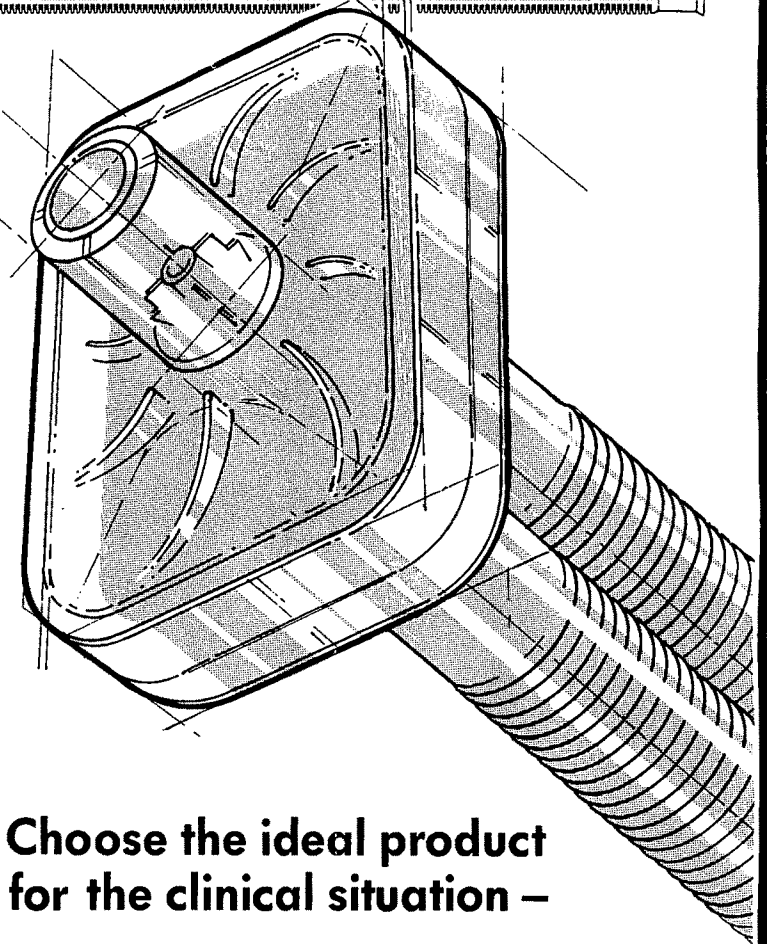
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Insertion of a partially inflated laryngeal mask airway

The technique used to insert the increasingly popular laryngeal mask is usually successful and well documented in print and on video, and I hesitate to suggest a modification.

I was enthusiastic from the outset, but there were still odd occasions when the smooth insertion of the mask was interrupted when either the tip buckled on the posterior pharyngeal wall or when placement over the laryngeal opening was imperfect. I have found that if the cuff is softly inflated in preparation, sufficient to remove all folds and indentations from the cuff, in my hands at any rate, insertion becomes even easier. The comparison with fitting

an inner-tube into a push-bike comes to mind when its partial inflation prevents the nipping or tucking which often occurs during this manoeuvre. In addition, with the correct position of the partially inflated laryngeal mask in the hypopharynx, confirmation is immediate with a flash of condensation in a transparent connector with the next expiration. Further air is then added to effect the typical inflation value.

South Cleveland Hospital,
Middlesbrough,
Cleveland

P.T.F. NEWMAN

The Woolley and Roe Case

The reassessment of the Woolley and Roe case by Hutter (*Anaesthesia* 1990; 45: 859–64) suggesting the arachnoiditis was caused by mineral acid was interesting, and, in my opinion, more plausible than the phenol contamination theory. Three clinical observations were cited in support of the mineral acid theory in the case of Cecil Roe, one of which related to an enlarging circular ulcer surrounding the point of the needle insertion, which continued to enlarge, reaching a size of 15 cm by the time of his death. Sadly, this communication from the family of Cecil Roe is not fully supported by the medical notes of the Sheffield Spinal Injury Unit, where Mr Roe was treated as both in and outpatient from 5 November 1947 until 28 August 1956 (he died in Chesterfield in October 1956). These notes are still available, and reveal that on admission on 5 November 1947 to this unit he had two 'pressure sores' one over the right greater trochanter and one on the lower part of the lumbar spine, both reported to be 'granulating and looking somewhat better'. Unfortunately, this is the only reference to a lumbar sore in the notes.

Examination in April 1948 only referred to 'some superficial sores to both buttocks'; there is no reference to a lumbar sore at this time, and the following month two attempts at lumbar puncture were performed, one at L₃₋₄ and one at L₁₋₂; both were reported as dry taps. Presumably these would not have been performed through possibly infected dead tissue, so it would be interesting to know the interspace used for the original spinal anaesthetic. Mr Roe was discharged as an inpatient in December 1948 but re-admitted in May 1949 because of 'a sacral sore developing over the past two months with spinous processes exposed'.

The notes continue to reveal problems with sacral sores until May 1950 when Mr Roe underwent exploratory neurosurgery in Birmingham. A laminectomy of T₁₀–L₁ was performed, and an arachnoid cyst at T₁₀₋₁₁ drained with the diagnosis of arachnoiditis confirmed. Postoperative stitch abscesses delayed recovery, but had healed by September when an intrathecal injection of 10 ml of absolute alcohol was performed in the low thoracic area over 4 minutes. Another spinal needle was inserted '2" further down' and a further 5 ml of absolute alcohol slowly injected. Despite these attempts to control spasm an obturator neurectomy was performed on 3 November 1950 followed in April 1951 by a further intrathecal injection of 5 ml of absolute alcohol at L₄₋₅ 'with great difficulty'. Sacral sores remained a problem for Mr Roe necessitating skin grafting in June 1951. Initially this healed well, but by

November had broken down again requiring excision of sacral bed sore with rotation flap from the right buttock. By March 1952 all skin areas were reported to be intact, and Mr Roe was discharged in August.

In August 1953 Mr Roe was readmitted for excision of a left ischial bed sore, and in June 1954 a further excision of this sore was done with rotation flap from the back of the thigh. By July this had healed completely, and he was discharged in October 1954 with 'intact buttocks and sacral area'. On 28 August 1956 an outpatient visit revealed a 'bed sore of the right buttock, present for 6 months', and this is the last record, apart from a note saying he was admitted to Chesterfield Royal Hospital on 15 October 1956 and died later that month.

This long, complicated saga illustrates that Mr Roe was prone to back sores, but apart from the admitting report, these were all sacral or pelvic. It is not correct therefore to assume that he had a lumbar ulcer from the spinal anaesthetic which had enlarged to 15 cm by his death. Almost certainly it was the bed sore of the right buttock noted in August 1956, 2 months prior to his death which the relatives recollect. What a pity further information is not given on the original lumbar 'pressure sore' which is not noted again.

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J.D. ALDERSON

A reply

Thank you for the opportunity to reply to Dr Alderson's letter. The information that there was ulceration around the site of the spinal needle insertion was given to me without prompting and quite spontaneously. It is unlikely that such an unusual phenomenon, the clinical significance of which was not apparent to the family, should have been recalled so emphatically if it had not been true. It is most probable that the 'pressure sore' over the lower point of the lumbar spine, which was noted on Cecil Roe's admission to the Sheffield Spinal Injury Unit, was in fact this ulcer which had been described by the family. It is apparent from the Sheffield notes that the ulcer subsequently healed and in this respect the family was mistaken. However, I believe that their original observations are still correct.

Queen's Medical Centre,
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C.D.D. HUTTER

Cardiac arrest following intramyometrial injection of prostaglandin E₂

We read with interest the report by Kilpatrick and Thorburn (*Anaesthesia* 1990; 45: 848–9) and wish to report a patient in whom intramyometrial injection of prostaglandin E₂ resulted in a cardiac arrest.

A 32-year-old primigravida delivered a live male infant vaginally. Bleeding from the vagina was seen at suturing of the episiotomy, but examination revealed no retained products. Blood loss was estimated at 600 ml. Haemoglobin was 11.2 g/dlitre and platelet count 155×10^9 /litre. Despite therapy with a syntocinon infusion, crystalloids and colloids, the bleeding continued. A decision was made to explore the uterus under a general anaesthetic. Plasma and blood were transfused before induction of anaesthesia, when the pulse rate was 100/minute and arterial blood pressure 120/65 mmHg. After pre-oxygenation of the lungs, anaesthesia was induced with thiopentone 150 mg and suxamethonium 100 mg to facilitate tracheal intubation, and maintained with 1% enflurane and 67% nitrous oxide in oxygen with 50- μ g boluses of fentanyl, and neuromuscular blockade with atracurium 25 mg. An internal jugular cannula was inserted which showed a CVP of 6 cmH₂O. The episiotomy sutures were removed. Examination revealed a bulky and hypotonic uterus without any retained products or deep cervical tears. The episiotomy was resutured and bimanual compression started to contract the uterus. The patient's pulse rate, arterial blood pressure and CVP remained stable during the procedure. One hour into the procedure, it became apparent that the bleeding was not being controlled and the obstetrician decided to inject prostaglandin directly into the myometrium. Prostaglandin E₂ in increments of 1 mg was injected blindly into the myometrium through the anterior abdominal wall. A few seconds after the third injection, the arterial pressure dropped from 120/65 mmHg to 80/40 mmHg and then became unrecordable, while the ECG trace showed a tachycardia of 150/minute rapidly

progressing to ectopic beats and ventricular fibrillation. Enflurane and nitrous oxide were stopped, the lungs were ventilated with 100% oxygen and external cardiac massage started. Resuscitation was in the form of DC shocks, atropine, adrenaline, lignocaine and more blood and plasma, resulting in a pulse rate of 150/minute and arterial pressure of 100/60 mmHg. The uterus was contracting well and the bleeding had lessened. The uterine cavity was packed and the syntocinon infusion continued. The patient's lungs were electively ventilated overnight in the ITU and her trachea extubated 18 hours after admission. She recovered without any residual neurological deficit and was discharged home after 9 days.

The dose of prostaglandin E₂ for intramyometrial use has not been described. In anaesthetised pregnant women, as little as 5 μ g intravenously has caused a decrease in arterial pressure to 70% and an increase in heart rate to 125% of control values within 60 seconds.¹ The rapidity with which hypotension and cardiac arrest followed the third injection of prostaglandin suggests that systemic absorption was responsible. The fact that the patient had increased sympathetic activity due to blood loss probably exaggerated the vasodilatory effects of the prostaglandin. There was no associated bronchospasm or skin rash to suggest an anaphylactic reaction to blood or drugs.

Northampton General Hospital,
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M.T. POPAT
N. SUPPIAH
J.B. WHITE

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Spinal needles, flow and dural puncture

I wish to comment on Dr Toomey's study of flow through spinal needles (*Anaesthesia* 1990; 45: 751–3). Although nominally 26 gauge, the two needles used may have been of different sizes. Apparently Braun follows US Standard, while Becton Dickinson use British Imperial Standard Wire Gauge. For 26-gauge needles the difference between the two is very small, but there is a case for quoting needle diameters in millimetres.¹ This, however, should not interfere with his conclusion.

Regional anaesthesia is very much an art, and I feel that scientific evaluations, out of context of the art, may be invalid. One key feature in the design of a spinal needle is its ability to tell the operator that dural puncture has occurred. If a group of needles are ranked first according to flow and then according to an observer's ability to recognise mock dural puncture, different rankings are obtained. Much of this is because of variation in hub design. Hubs vary from manufacturer to manufacturer, and even from gauge to gauge for a single manufacturer. A needle with a slow flow but a communicative hub will give a quicker report than a fast needle with a secretive hub.² If Dr Toomey 'noticed some technical difficulties', for me this is a far more significant finding than a difference in flows.

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Leeds LS9 7TF

G. LYONS

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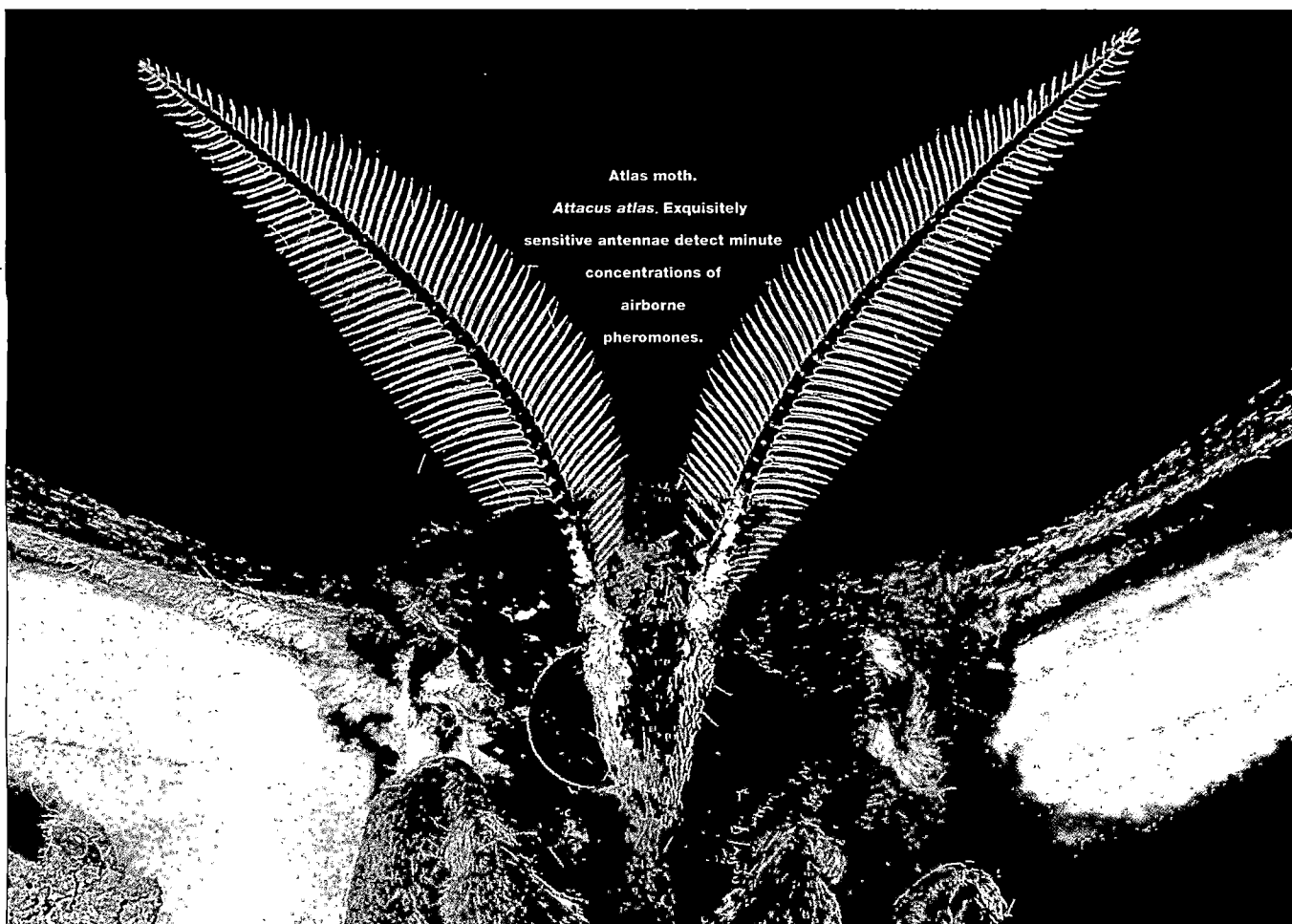
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A reply

Thank you for the opportunity to reply to Dr Lyon's letter.

The 'technical difficulties' considered to be so significant were those referred to in the subsequent sentences of the article noting the prolonged time taken for cerebrospinal fluid (CSF) to appear in the hub of the needle. Regional anaesthesia may be an art and there may be an art in placing a needle in the subarachnoid space, but most practitioners still rely on the appearance of CSF to confirm placement when using a needle of this calibre. Experienced practitioners have been misled when using this needle because of the long interval before CSF is seen. Science has been applied to identify the possible cause of this problem and has led to improvement in the standard of equipment in the past.¹

The problem of communicative hubs was considered. Both needles have transparent hubs, that of the Becton Dickinson needle being lightly tinted. In a pilot study, split



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Maintenance: Usually 0.1 to 0.2mg/kg/min (6 to 12mg/kg/hr). Continuous infusion may require slightly higher rates 'or 10 to 20 minutes after induction. The infusion can be used undiluted or diluted with 5% Dextrose (intravenous infusion BP). Alternatively repeat bolus injections of 25 to 50mg (2.5 to 5.0ml) may be administered according to clinical need.



Paediatric Use: No experience in children or in mothers who are breast feeding.

Contraindications: Allergy to 'Diprivan'.

Precautions: Ampoules and vials should be inspected before use for particulate matter and discolouration. Aseptic technique should be followed throughout its handling; administration commencing without delay once drawn into the syringe or giving set. Discard any unused 'Diprivan' after single patient use. Do not mix prior to administration with other agents or infusion fluids other than Dextrose 5%, such dilutions should be prepared immediately before administration. Hypotension and transient apnoea may occur during induction. Occasionally, hypotension may require use of i.v. fluids and a lower rate of administration during maintenance. Apply caution in cardiac, respiratory, renal or hepatic impairment; epilepsy; in hypovolaemic or debilitated patients; and in disorders of fat metabolism or conditions where lipid emulsions should be used cautiously. Bradycardia may occur due to the lack of vagolytic activity of 'Diprivan' and administration of an anticholinergic should be considered particularly if vagal

tone is dominant. Do not use in pregnancy except for termination. Discharge after general anaesthesia - allow adequate time for full recovery.

Side effects: Epileptiform movements, including convulsions and opisthotonus, have occurred rarely in a temporal relationship to 'Diprivan'. Nausea, vomiting and headache in a small proportion of patients. Very rarely, clinical features of anaphylaxis, which may include bronchospasm and erythema accompanied by hypotension, have been reported with 'Diprivan'. Pain on injection in a proportion of patients. Discolouration of urine, venous sequelae and fever are rare. Minimal evidence of excitation on induction.

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times of the first appearance of fluid in the hub and times of the first drops falling from the hub were obtained. However, there was considerable observer variation in the first timing and much more consistency in the second measurement, hence this timing was used to reduce any error produced by poor visibility in the hubs.

There are two standards for describing the diameters of needles. The Braun 120 mm Spinocan was compared with the Becton Dickinson needle, because it was the needle that we had used previously for spinal anaesthesia. However, flows through 90 mm 26-gauge Braun needles and 90 mm

26-gauge Becton Dickinson needles have been shown to be identical.¹

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P.J. TOOMEY

Reference

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Glove-wearing by anaesthetists

I read with interest the recent article by Harrison *et al.* (*Anaesthesia* 1990; **45**: 831-3) entitled 'Blood contamination of anaesthetic and related staff'. Of particular interest was the table documenting the glove-wearing habits of the Cardiff anaesthetic department which seemed to be representative of the glove-wearing habits of the various departments I have worked in around this country. Having recently worked in the United States, where the prevalence of glove-wearing by the anaesthetic staff appeared to be very high, I thought it might be of some interest to document this practice in a large American teaching hospital. On my latest visit back to the United States I canvassed some of my former colleagues in order to compare their habits with those published from Cardiff.

I interviewed 74 'anaesthetic providers' who work within the University of Maryland Medical System in Baltimore; 34 staff anesthesiologists, 24 resident anesthesiologists and 16 nurse anesthetists, all of whom regularly placed tracheal tubes, peripheral venous, arterial and central venous lines. Table 1 shows the level of glove-wearing for these four procedures. Table 2 lists the reasons given for not wearing

gloves. Both tables contain the corresponding data from Cardiff. The high level of glove-wearing in Baltimore (and the United States in general) could be explained by peer group pressure (nonglove-wearing frequently draws comment from other health care workers), higher levels of infectivity in American inner city populations (University of Maryland Medical System accident department sees a 7% HIV positivity rate in its admissions) or just a greater awareness by health care workers about the need to protect themselves. I believe that the higher rate of glove-wearing and better level of hepatitis B immunisation reflects a more up-to-date attitude in self protection in the health care field. This would appear to be supported by the data in Table 1 showing that staff anesthesiologists have lower rates of glove-wearing and hepatitis B protection than their juniors. The explanation for so many respondents finding difficulty with adhesive tape (Table 2) is that most tracheal tubes in the University of Maryland Medical System are secured using tape, and 87% of the anesthesiologists use gloves to intubate.

Wearing gloves does not confer protection against most

Table 1. Percentages and numbers of those routinely wearing gloves.

	University of Maryland Medical System at Baltimore, USA				Cardiff
	Total	Staff	Resident	Nurse anesthetist	Total (Harrison <i>et al.</i>)
Oral/nasal tracheal intubation	87% (65)	82% (28)	92% (22)	94% (15)	9% (7)
Peripheral cannulation	55% (41)	44% (15)	62% (15)	69% (11)	8% (6)
(those wearing gloves not as a routine but more than 60% of the time)	11% (8)	6% (2)	17% (4)	12% (2)	
Arterial cannulation	84% (62)	73% (25)	96% (23)	87% (14)	63% (47)
Central cannulation	100% (74)	100% (34)	100% (24)	100% (16)	89% (67)
Hepatitis B immunisation	84% (62)	82% (28)	92% (22)	75% (12)	71% (53)

Table 2. Reasons given by those not wearing gloves as a routine.

Reasons given	Baltimore	Cardiff (Harrison <i>et al.</i>)
Difficulty using adhesive tape	38	3
Loss of sensitivity	29	42
Habit	22	25
Low chance of infection	16	5
Convenience	16	20
Ignorance and laziness	8	10
Discomfort of wearing gloves	6	15
No protection against needle or eye contamination	5	7

needlestick injuries, neither is every patient a source of infection, but with constantly growing numbers of unidentified HIV-positive patients on this side of the Atlantic, I do not think that anyone can ignore the protection offered by gloves. Would anyone not wear

gloves when anaesthetising a patient identified as being HIV or hepatitis B antibody positive?

Bristol Royal Infirmary,
Bristol BS2 8HW

P.J. MILLS

Withdrawal syndrome after propofol

In their case report of a withdrawal syndrome following propofol infusion (*Anaesthesia* 1990; 45: 741-2), Dr Au and his colleagues provide little conclusive evidence for the implication of propofol and draw perhaps unwarranted conclusions.

As the authors say, convulsions may complicate cardiopulmonary bypass with deep hypothermia and circulatory arrest. Additionally in the case described, the cerebral circulation may have been compromised pre-operatively by the aortic dissection, which extended as far proximally as the ascending aorta. Given these possible pre- and peri-operative causes for convulsions, propofol's anticonvulsant effect may well have suppressed epileptic activity until its withdrawal. While the patient apparently regained mental function 12 hours postoperatively, the authors report increasing agitation which may have been indicative of a developing mental problem. In our experience, propofol is an extremely effective anti-convulsant agent. We have successfully used it in status epilepticus on at least six occasions, including three cases where successful anticonvulsant effect was confirmed by EEG. Several of the cases, which included encephalitis, metabolic encephalopathy, head injury and cerebral hypoxia following cardiac arrest, had proved resistant to conventional therapy. A case report describing two of these cases has been accepted for publication in *Anaesthesia*.

This ITU has used propofol fairly widely for sedation with over 100 patients receiving it for between one and 30 days. We have not seen any evidence of a withdrawal syndrome or convulsions associated with its use. This case appears to have too many other factors involved to draw any conclusions or make any recommendations.

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I.S. GRANT
M. WORSLEY

A reply

We thank Drs Grant and Worsley for their interest and are grateful for the opportunity to reply.

In our opinion, complications of aortic dissection and/or cardiopulmonary bypass with deep hypothermia and circulatory arrest (CPB-DH-CA) cannot explain our patient's symptomatology. Firstly, it is debatable whether

the left lower limb monoplegia was due to cerebral or spinal ischaemia. Moreover, the subsequent clinical course mitigates against a simple ischaemic stroke. Secondly, although convulsions can complicate CPB-DH-CA, patients so affected typically manifest delayed waking as the first sign of serious neurological insult.¹ This was not the case in our patient. Thirdly, seizures complicating CPB-DH-CA are typically transient and followed by uneventful convalescence,^{2,3} unlike our patient to whom an organic brain syndrome persisted for a further week before recovery.

We congratulate Drs Grant and Worsley on their successful use of propofol in treating status epilepticus. However, this in no way implies that withdrawal seizures cannot occur with discontinuation of this drug; intravenous diazepam is likewise an anti-epileptic agent and is also known to cause withdrawal seizures in dependent patients.⁴ It is, of course, unrealistic to expect a case report to provide conclusive evidence and cast-iron recommendations. We believe, however, that the temporal association of propofol discontinuation and the development of the classical symptomatology of a general depressant withdrawal syndrome seriously implicate propofol in a causative role. Furthermore, although confounding factors may be present, we feel that it would be imprudent indeed to dismiss outright the possibility of drug dependency and its attendant hazards after prolonged infusions of propofol.

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J. AU
D.H.T. SCOTT
W.S. WALKER

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Forewarning patients of sexual arousal following anaesthesia

Boheimer and Thomas (*Anaesthesia* 1990; 45: 699) advised that 'susceptible' patients should be forewarned of the possibility of postoperative sexual fantasies and amorous behaviour before administering midazolam or propofol. We consider that this is not a practical suggestion. The pre-

operative visit is a very important moment for establishing a rapport with our patients. To discuss the risks and advantages of different anaesthetic techniques is necessary, but pointing out problems whose incidence and importance has not yet been established is unwarranted. To talk about

sexual matters without inducing anxiety or uneasiness would be desirable, but in our cultural environment difficult to achieve.

We are aware of only one female patient who was distressed by sexual phantasies in the recovery period. All our other patients who recalled sexual dreams or amorous behaviour were satisfied, and some even enthusiastic, with the anaesthetic that consisted of midazolam premedication and total intravenous anaesthesia with propofol. We therefore prefer to forewarn staff rather than patients of the above-mentioned arousal phenomena. To our mind the problem with these drugs is not so much sexual phantasy or amorous behaviour, but the documented association of midazolam^{1,2} and propofol³ with allegation of impropriety. Although we share the concern of Boheimer and Thomas as far as the postoperative side effects are concerned, we

suggest that only if a third party cannot be made present, that the use of midazolam and propofol should be re-considered.

University of Basel/Kantonsspital,
CH-4031, Basel,
Switzerland

H.G. SCHAEFER
S.C.U. MARSCH

References

1. DUNDEE JW. Unpleasant sequelae of benzodiazepine sedation. *Anaesthesia* 1990; **45**: 336.
2. DUNDEE JW. Complaints of sexual fantasies following benzodiazepine sedation in women. *Anesthesiology* 1990; **73**: A27.
3. SCHAEFER H-G, MARSCH SCU. An unusual emergence after total intravenous anaesthesia. *Anaesthesia* 1989; **44**: 928-9.

Hazard Notice

**Oxylitre emergency/rescue medical gas manifold assembly:
possible risk of nonreturn valve fracture HC (Hazard) (90)
43**

A size 18 mm nonreturn valve fractured during cylinder changeover causing the valve interior to be ejected under full cylinder pressure. Nonreturn valves now manufactured measure 20.8 mm and the manufacturers recommend replacement of their earlier smaller valves.

Vi-Tal Hospital Products Ltd HC (Hazard) (90) 44

Readers should note that this company, which manufactures Vi-flow syringe pumps, ceased trading on 22 October 1990. Problems securing service and repair of equipment and obtaining dedicated disposables are to be anticipated. Further information will be issued when available.

Safety Action Bulletin

Pulse oximeter: noncompatibility of sensors (SAB (90) 79)

A sensor supplied by Physio Control for use with their Lifestat pulse oximeter can be plugged into Ohmeda pulse oximeter models 3700, 3710 and 3740. Should this occur, the sensor rapidly overheats and there is the possibility of the patients receiving burns. Both manufacturers are aware of the problems. Only those sensors recommended as being compatible with their equipment should be used.

Errata

Anaesthesia, 1990, Volume 45, page 887

The interpretation of results by doctor technicians

M. P. D. HEINING

The third sentence in the third paragraph of this letter should read: 'Sample heparinisation by filling the syringe deadspace with intravenous sodium heparin 1000 IU/ml ...' and not sodium heparin 1000 ml.

Anaesthesia, 1990, Volume 45, pages 946–948

Life-threatening complications during anaesthesia in a patient with a ventriculo-atrial shunt and pulmonary hypertension

P. J. BUTLER, R. A. WHEELER AND P. M. SPARGO

The dose of fentanyl administered to this patient was 1.0 $\mu\text{g/kg}$ and not 0.1 mg/kg as printed in the penultimate paragraph before the discussion.

pH-adjustment and discomfort caused by the intradermal injection of lignocaine

A. J. MARTIN

Owing to a printer's error two columns in Table 5 were inappropriately headed. The correct version of the Table is printed below.

Table 5. Between-group differences in infiltration pain scores and comparisons of modified and unmodified solutions.

	Pain score		Comparison using Mann–Whitney <i>U</i> test
	Mean	Median	
1% plain lignocaine	1.1	1	1% lignocaine as compared with 1% lignocaine with sodium bicarbonate ($p < 0.02$)
1% lignocaine with sodium bicarbonate	0.6	0.5	
1% lignocaine with 1 : 200 000 adrenaline	1.5	1	1% lignocaine with 1 : 200 000 adrenaline compared with 1% lignocaine with adrenaline and sodium bicarbonate ($p < 0.001$)
1% lignocaine with adrenaline and sodium bicarbonate	0.5	0	

Under the Acknowledgments heading Dr J. Kinsella was thanked for his help with the method of randomisation in the study. This should have been Dr T. Kinsella.

Noncardiogenic pulmonary oedema after attempted suicide by hanging

S. MANTHA AND S. M. RAO

In Table 1 in this letter, PEEP values given as 10 and 6 should be 1.0 and 0.6 respectively.



Association of Anaesthetists of Great Britain and Ireland

Annual Report of Council 1989–1990

1. Membership of the Association (as at 30.7.90)

The total Membership of the Association is 5757 (5617).

Honorary	36	(34)
Ordinary	2092	(2073)
Trainee	2117	(2064)
Corresponding	33	(33)
Overseas	868	(903)
Retired	547	(488)
Senior	9	(10)
Associate	13	(12)
Joint	42	

(Last year's figures in brackets).

There were 35 resignations during the year.

Deaths

Council records with regret the death of the following members:

Bhayat, R.E. (*South Africa*), Burns, T.H.S. (*London*), Cronin, M. (*London*), Dalas, S. (*Birmingham*), Davidson, J.T. (*Israel*), Davidson, L.D. (*Aberdeen*), Grenville, H. (*London*), Griffin, K. (address unknown), Hain, W.R. (*Nottingham*), Houldsworth, B. (*Barnsley*), Knowles, G.S.A. (*Suffolk*), MacDonald T.R. (*Australia*), Powell, K.J. (*Devon*), Robertson, J.D. (*Edinburgh*), Rollason, W.N. (*Aberdeen*), Wood-Smith, F.G. (*Kent*).

2. Honorary Membership

Council recommended for election to Honorary Membership:

Dr T.B. Boulton; Dr R.J. Maxwell; Professor O. Mayrhofer; Professor H. Ruben.

3. Pask Certificate of Honour

Council awarded Pask Certificates of Honour to:

Dr L. Kaufman, Consultant, London, Collator of anaesthetic literature section in *Anaesthesia*; Dr Wendy E. Scott, Consultant, Milton Keynes, Association Exhibition Stand Co-ordinator; Mr B.R. Sugg, Bsc (Eng), Secretary, British Anaesthetic and Respiratory Equipment Manufacturers Association; Dr R.S. Vaughan, Consultant, Cardiff, Association Technical Exhibitions Co-ordinator.

4. Annual Scientific Meeting 1989

The Annual Scientific Meeting was held at the Taliesin Centre, University College, Swansea, on Thursday and Friday 14–15

September 1989. There were 521 registrants. A stimulating scientific programme, complemented by a delightful social programme, was organised by Dr Patricia Steane, Chairman, Local Organising Committee, Dr Rosemary Mason, Scientific Programme Organiser, and their colleagues from the Department of Anaesthesia, Swansea. The John Snow Lecture, 'The Good of the Child', was delivered by Baroness Warnock, DBE, of Girton College, Cambridge. The technical exhibition, with 93 stands, was larger than at any previous Association meeting.

The Annual Dinner was held at the Brangwyn, Hall, Guildhall, Swansea. Mr Alun Williams OBE, BBC Radio Wales gave the toast 'The Association'. Other distinguished guests included Baroness Warnock, Councillor Mrs L.J. Aldran, Lord Mayor of Swansea, Professor Sir Gordon Robson CBE, Professor M. Rosen and Professor J.A. Beardmore.

5. Annual General Meeting 1989

The Annual General Meeting was held at the Taliesin Centre, University College, Swansea, on Friday 15 September 1989. The President, Dr M.M. Burrows, presided and 182 members attended.

Honorary Membership was conferred on Dr M.P. Coplans (citation, Professor M.D. Vickers), Dr M.J. Johnstone (citation, Dr P. Morris), Professor J.P. Payne (citation, Professor A.P. Adams). Dr Carlos Parsloe was elected to Honorary Membership in absentia.

Pask Certificates of Honour were awarded to Dr P.J. Butler, Leicester, Dr E. Egan, Tanzania, the late Dr D.S. Savage, Glasgow, and Dr M.J. Mowbray, Nottingham.

The Minutes of the Meeting have been circulated to Members.

6. Linkman Conference 1989

The 14th Annual Conference of Linkmen was held at the Taliesin Centre, University College, Swansea, on Wednesday 13 September 1989. The President was in the Chair and 192 Linkmen attended. The principal topics addressed included the Bevan Report/Efficiency of Theatre Service (Dr E.B. Lewis), medical indemnity insurance (Dr J.A. Wall, MDU), pre-anaesthetic check procedures (Professor A.P. Adams), meritorious service awards (Professor Sir Gordon Robson CBE) and the Government white paper (Mr D. Nicholl CBE, Chief Executive NHS).

A detailed report of the Conference was published in *Anaesthesia* (1990; 45: 263–9).

7. Postgraduate Study Day 1989

This meeting, organised jointly by the College of Anaesthetists and the Association, was held on Saturday 21 October 1990 at the

Royal College of Surgeons of England, London. Despite a smaller attendance (200) the success of this meeting was maintained. As in previous years, the format enabled registrants to attend six lectures of their choice from the total of 18 lectures delivered.

8. Winter Scientific Meeting 1990

The third annual Winter Scientific Meeting of the Association was held at the Royal College of Surgeons of England, London, on 5-6 January 1990. The excellent scientific programme, prepared by Dr W.S. Nimmo, assisted by Dr J.A.W. Wildsmith and Dr J.F. Nunn, was attended by 282 registrants.

The highlight of the meeting was the inaugural BOC Health Care Lecture, 'The Air We Breathe', delivered by Dr J.F. Nunn, Vice President. The Association is grateful to BOC Health Care for their support of this prestigious annual guest lecture.

The Winter Dinner was held at the Waldorf Hotel, London, on Friday 5 January 1990. The Toast, 'The Association' was given by Mr L.J. Blom-Cooper QC, Chairman, Press Council. Other distinguished guests included Sir Donald Acheson, Professor Sir Herbert Duthie, Sir George Pinker KCVO, Professor Sir Gordon Robson CBE, Sir Anthony Grabham, Professor M. Rosen CBE, Mr D.K. Nichol CBE, Professor T.C. Gray CBE, Professor J.D. Chisholm and Professor J.B. Howell.

9. Junior Anaesthetists Group Annual Scientific Meeting 1990

This successful meeting, hosted by the Glasgow Divisions of Anaesthesia, was held at Jordanhill College, Glasgow, 4-6 April 1990. The attendance, 333 registrants, was the largest ever. The meeting was organised by Dr D. Dutton, Chairman, Dr M.D. Inglis, Dr I. McMenamin, Dr D.L. Paul and Dr Joyce Reid.

The scientific programme included sessions devoted to intensive care, paediatric anaesthesia and intensive care, current issues in obstetric anaesthesia, control of blood pressure, the lung, management of intractable pain and management of the burned patient.

The Pinkerton Lecture, entitled, 'What's New at the Neuromuscular Junction?' was delivered by Professor W.C. Bowman, Professor of Pharmacology and Vice Principal, University of Strathclyde.

The President's Medal for the Junior Anaesthetists Annual Prize Competition was awarded to Dr S. Ridley, Senior Registrar, Glasgow, for his paper entitled 'Long-term outcome from ITU'. The second prize was shared by Dr H.E. Jones, London and Dr I.H. Lewis, Winchester.

10. Other Meetings

- (i) *First International Conference on Anaesthesia and Critical Care in Disasters and War*. This meeting was held at the Royal College of Surgeons of England, London, 18-21 July 1990, under the patronage of the Surgeon General, the President, College of Anaesthetists, and the President, Association of Anaesthetists. There were 254 registrants, many of whom were from the Continent of Europe and North America. The Joint Organising Committee, Convenor, Air Commodore C.A.B. McLaren, compiled an interesting scientific programme. The topics addressed included trauma, oxygen, anaesthesia in the field, resuscitation and casualty transport. Dr Gillian Hanson, London, delivered the guest lecture entitled 'Controversies in Intensive Care'.
- (ii) *8th European Congress of Anaesthesiology*. The 8th European Congress, hosted by the Polish Society of Anaesthesiology and Intensive Therapy, will be held in the Palace of Culture and Science, Warsaw, 9-15 September 1990. The General Assembly of the European Section, WFSA, will take place at the same venue on Thursday, 13 September. It is anticipated that the Congress will be well supported by members of the Association.

To mark the occasion the Association will present a badge of office to the Polish Society of Anaesthesiology and Intensive Therapy at a reception to be held at the British Embassy, Warsaw, on the evening of Tuesday, 11 September.

11. Council

Throughout the year October 1989 to September 1990 both Council and the Advisory Committee met on five occasions. The attendance of Officers and Elected Members at Council and Advisory Committee is detailed in Appendix A, together with the membership of sub-committees and working parties.

Council warmly congratulated Professor M. Rosen, Immediate Past President of the Association and President of the College of Anaesthetists, on being appointed CBE in the New Year's Honours List. Professor Rosen will retire as Immediate Past President. Council gratefully acknowledges the inestimable value of his many innovative contributions to the Association during the 18 years since his election to Council. The benefit of his wise counsel will be retained, since Professor Rosen, in his capacity as President of the College of Anaesthetists, will continue to serve as a co-opted Member of Council.

On completion of two years as Vice President, Dr J.F. Nunn also retires from Council. His valuable contribution to the activities of the Education and Research Committee and in supporting Dr J.A.W. Wildsmith, Seminars Co-ordinator, are deeply appreciated.

Dr J.N. Lunn, having served two years as Joint Editor, and eight years as Editor of *Anaesthesia* has completed the maximum term of office and will retire with effect from the 1990 Annual General Meeting. The Association and all anaesthetists are indebted to Dr Lunn. His continuous commitment to excellence and the highest standards have ensured that *Anaesthesia* is a leading journal for anaesthesia worldwide. In addition, Council recognises his much valued contribution as a long serving Officer of the Association.

Council is particularly pleased to support the recommendation of the Editorial Board that Dr M. Morgan, London, be nominated for election as Editor with effect from the forthcoming Annual General Meeting.

Dr D.S. Arthur, Dr J. Edmonds-Seal and Dr J.A.W. Wildsmith will retire from Council having served as Elected Members. Their respective contributions to the work of the Association are gratefully acknowledged. In particular, Council is grateful to Dr Edmonds-Seal for his expert Chairmanship of the Anaesthetics Sub-Committee, CCSC, and to Dr Wildsmith who has served with distinction as Chairman of the Education and Research Committee and Seminars Co-ordinator.

Council warmly records its appreciation of the services of the Association's secretarial staff, Miss Ann Muir, Administrative Secretary and her assistant Mrs Betty Tyler, Ms Pat Plant, Financial Secretary, assisted by Miss Lyn Hunt, Mrs Lesley Ogg, BOC Educational Co-ordinator, Mrs Mary Richards, Minute Secretary, and Dr Audrey Eccles ICI Archivist/Librarian. Recently Mrs Richards resigned to set up a home-based computing service and we welcome Ms Sally Jenner who has taken up the vacant post.

Mr E. Warburton, Honorary Financial Advisor, continues to give outstanding support and sound advice on all financial and legal matters. Council is deeply grateful to him.

In December 1989 the following resolution was agreed by Council. 'Council of the Association of Anaesthetists recognises the need for the College of Anaesthetists to extend its development by seeking legal identity through a separate charter, by seeking Royal status and by the acquisition of independent premises. Council of the Association is unanimous in expressing its strong support to the College of Anaesthetists in pursuing these aims.'

This resolution received the unanimous support of members attending the Winter Scientific Meeting.

A joint Association/College Working Group was convened in response to an invitation to comment on the issues raised in the terms of reference of the Department of Health (DOH) Working Party on Dental Anaesthesia (Chairman: Professor D. Poswillo). The Group stressed that standards of anaesthesia practised in dental surgeries should be the same as in hospital. Patient monitoring requirements, levels of assistance and training in sedation techniques and resuscitation were also addressed. The view was expressed that dentists should no longer receive training in the practice of anaesthesia. The report of the DOH Working Party is awaited.

Dr P. Morris, Vice President, prepared a discussion paper in response to concerns expressed by members of problems associated with training ambulance personnel for an extended role. Subsequently, a Working Party, Chairman: Dr P.J.F. Baskett,

President Elect, was set up to examine the role of the anaesthetist in the emergency service, including the training of ambulance personnel. Membership includes two consultants in A&E Medicine, a College representative and a consultant anaesthetist with a special interest in A&E Medicine. The Working Party has met three times and the preparation of a report to Council is well advanced.

The 60th anniversary of the founding of the Association will take place in 1992. A small Steering Committee, Chairman, Dr P.J.F. Baskett, has been set up to make appropriate arrangements.

Council was pleased to note the first Report of the National Confidential Enquiry into Peri-Operative Deaths, 1989, (Campling E.A., Devlin, H.B. and Lunn, J.N., 1990). The Report reviews the surgical and anaesthetic care of children in hospital.

The West Midlands Regional Health Authority's recently published 'Planning Guide No. 104, Department of Anaesthesia', which offers guidance to health authorities and anaesthetists on the design of existing and projected departments. In commending this publication to members, Council wishes to thank Dr P.V. Scott, Consultant Anaesthetist, Redditch, for his valuable contribution to this document and for arranging that the Association was given an opportunity to comment on the document at its draft stage.

The Association has been represented by the Assistant Honorary Secretary on a working party of the British Society of Gastroenterology considering safety and monitoring in endoscopy. Some progress has been made in improving safety although it is apparent that much work remains to be done. A report will be presented to the Linkman Conference.

12. Association of Anaesthetists Standing Committee, Republic of Ireland

The Standing Committee at the Royal College of Surgeons in Ireland met in February 1990. Dr M.M. Burrows, President, Dr W.R. MacRae, Honorary Treasurer, and Dr W.L.M. Baird, Honorary Secretary, attended.

Efforts to achieve the implementation of recommendations contained in the Standing Committee's publication, 'Anaesthesia in Ireland—The Provision of a Safe Service', continues. A dialogue on professional fees is being maintained between representatives of the Standing Committee and the Voluntary Health Insurance board.

A draft extended constitution, setting out proposals on electoral procedure, has been prepared and will be considered by Council.

13. Linkman Organisation

Throughout the year Dr J.E. Charlton, Assistant Honorary Secretary, has kept members well informed of Council's activities and in turn maintained the valued links between members and Council. Three Linkmen letters were circulated drawing attention to the major topics discussed in Council.

Appendix C lists current Linkmen and the hospital(s) or health authority which they represent.

14. Seminars and Workshops at Bedford Square

Throughout the year 35 seminars were held, covering a wide range of topics. They were virtually always over-subscribed, a good testimony to their success. Members are requested to appreciate that accommodation is limited and to make early application. Some seminars are to be repeated.

Council is deeply grateful to Dr J.A.W. Wildsmith, Seminar's Co-ordinator, the members of the Organising Committee, Dr J.F. Nunn and Sqn Cdr S.B. Merrill, and Mrs Lesley Ogg, BOC Educational Co-ordinator, for their hard work in ensuring the continuing success of the seminars programme.

Council is particularly pleased to announce that Professor A.R. Aitkenhead, Nottingham, has agreed to take over the duties of Seminars Co-ordinator at the start of the 1990/91 session.

15. Education and Research Committee

The Committee (Chairman Dr J.A.W. Wildsmith) met three times during the year and continued its work supervising the

Association's post-graduate meetings and seminars as well as adjudicating applications for research and travel grants. In regard to the latter, the Committee has produced a new document entitled, 'Educational Awards', to guide members when making application for financial support. This document is now available from Bedford Square.

Association Research Fellowship, 1990-92

Six applications were received. The Appointment Committee recommended that no appointment be made and the post re-advertised early in 1991.

Research Grants

Council approved the following applications on the recommendation of the Education and Research Committee: Dr J. Sear (*Oxford*) £2624 to study interaction of general anaesthetics with plasma proteins using ¹H-NMR. Dr A.P. Baranowski (*London*) £4698 to study neuropeptides in neuropathology. Dr M. Reeder (*Oxford*) £3396 to study peri-operative respiratory, haemodynamic and electrocardiographic changes in patients) undergoing elective abdominal vascular surgery.

Three other requests were reviewed but not recommended to Council.

Travel Grants

Council approved the following applications for travel grants recommended by the Committee: Dr J.P. Nolan (*Bristol*) £500 in support of a visit to trauma centres in eastern USA. Dr M.A. Garrioch (*Glasgow*) £500 in support of a visit to trauma centres in Washington DC and Baltimore. Dr A.J. Coe (*Liverpool*) in support of a visit to the Pain Relief Clinic, Seattle.

Baxter Healthcare Travelling Fellowship, 1989

This Fellowship was awarded to Dr I.W. McConachie, Manchester, to visit the R. Adams Cowley MD, Shock Trauma Centre, Baltimore, to study the factors influencing the choice of intravenous fluids for resuscitation and during anaesthesia after major trauma.

The Committee encourages more members to apply for this valuable Fellowship.

Undergraduate Essay Prize, 1990

An Undergraduate Essay Prize was awarded to Mr J.R. Ashley (*University of Leeds*) for his essay entitled, 'Problems of treatment of post-herpetic neuralgia'.

The Association acknowledges with gratitude the assistance given by experts in reviewing applications throughout the year.

Future Meetings

The calendar of future, national and international meetings is detailed in Appendix B.

16. Safety Committee

The Committee (Chairman Professor P. Hutton) has met on three occasions and has considered a wide range of topics concerning all aspects of safety in anaesthesia. Council has set up a Working Party to make recommendations on standards of safe practice in anaesthesia which will report next year.

The Committee considered mechanisms for informing the profession of the withdrawal or discontinuation of production of drugs. Members have complained that companies gave insufficient notice about the withdrawal of certain drugs or anaesthetic agents. The Association has made strong representations to the manufacturers to improve consultation mechanisms.

Members have expressed concern over Safety Action Bulletin (SAB [89] 78) concerning breathing systems. The Safety Committee has been able to direct expert advice from members to the appropriate quarter and as a result a new SAB is to be issued shortly. Potential problems may exist with regard to product liability following implementation of an EEC Directive (85/374) in July 1988. An explanatory booklet is available from the Medical Defence Union.

It has been noted that doctors from EEC countries have the right to employment in the UK without the need to pass any test of proficiency of written or spoken English. This may lead to problems with comprehension and communication which have clear implications for patient safety. Members are reminded that it is their responsibility to ensure that doctors from EEC countries have adequate written and spoken English before employment, especially in locum posts.

The Committee has continued to press for adequate representation and funding of British anaesthetic interests on all committees concerned with British, European and international standards. The Committee believes that British anaesthetists should play a full part in formulating these standards, and that any trend for standards to become industry-led should be resisted.

17. International Relations Committee

The Committee (Chairman Dr S.M. Lyons) met on two occasions during the year. They have continued their contacts with countries requesting assistance on advice in the teaching and practice of anaesthesia. Two-way exchanges of personnel between the UK and overseas posts continues.

The Committee has maintained contacts with the British Council, the Overseas Development Association and WFSA in the pursuit of similar aims. The establishment of an equivalent Committee to the International Relations Committee by the American Society of Anesthesiologists is of major interest.

The Committee continues to review its areas of interests. Opportunities are now occurring in eastern Europe to arrange exchanges and take part in teaching and refresher courses. Council has supported the principle of fostering such opportunities.

The Committee proposes to set up a database to co-ordinate information about overseas posts, applications and applicants, and to keep reports from previous holders of such posts, especially those in the developing world. It is hoped that this will be in operation in late 1990.

18. Editorial Board

The Board met on two occasions in 1989/90 under the chairmanship of the President. Dr J.N. Lunn has resigned as Editor with effect from September 1990 having completed eight years as Editor preceded by two years as Joint Editor. Dr M. Morgan, London, is nominated for election as his successor. Dr Rosemary A. Mason, Swansea, was appointed as Assistant Editor; she commenced duties on 1 June 1990.

It was agreed that, as a result of a postal survey of members and the experimental withdrawal of the feature 'Anaesthetic Literature', publication of this should cease from 1 January 1991. The services of Dr L. Kaufman over an extended period (25 years) for this section of the Journal was recognised by the Board. The Association is grateful to Dr Kaufman for his sustained and conscientious service which he has rendered to the Journal for this long period of time.

Anaesthesia News is no longer a responsibility of the Editorial Board following the establishment of a separate Management Board composed of the Editor, Dr J.N. Horton, the Officers of the Association and one Elected Member. A libel action against *Anaesthesia News* consequent on an article was avoided by a publication of an apology and payment of damages to Professor J.S. Robinson.

19. Finance Committee

The Committee (Chairman Dr M.M. Burrows, President) met twice during the year. The continuing high level of activities had been contained financially, but the Committee were well aware

that, because of the increasing number of members attending, moving both the Annual and Winter Scientific meetings into commercially operated conference facilities would have considerable financial implications. The contribution of funding towards the newly appointed Senior Lecturer in Intensive Care in the University of Birmingham was also taken into account when it was decided to recommend that the full ordinary subscription from July 1991 should be £100 with increases, pro rata, in other subscription rates.

The audited accounts have been circulated to members.

20. Private Practise Committee

The Committee (Chairman Dr R.M. Weller) has met on four occasions.

A guideline for fees for 1990 based on the familiar eight categories of operation was produced. This was planned to cover only one year because of the rapidly changing situation within the independent sector.

The Committee responded to the BMA's publication, 'Private Consultant Work, BMA Guidelines, 1989'. The Committee was most unhappy with this document and through the Anaesthetics Sub-Committee, CCSC, expressed a strong desire to be involved actively with the production of the revised version, to be published at the end of 1990. The Committee felt that recommendations from a professional organisation on grading of operations, and fees to be charged, were to be encouraged. Attempts to agree on the revision of this document are being undertaken at present.

The Committee wrote to over 15 provident societies and/or insurance companies involved with paying benefits in the independent sector. Replies were hard to obtain. The companies were informed of the Association's advice to its members about fees, of the need to separate surgical and anaesthetic benefits and of the Association's views on the payment of non-accredited specialists.

A further matter considered was the percentage of the total fee where it exists, or of the surgical benefit where the benefits are separated, that the anaesthetist should receive. As before, it was felt that half the surgeon's fee, or a third of the total, was appropriate.

The Committee tried to persuade the provident societies to abide by their own rules and only pay benefits to accredited anaesthetists. This is not yet resolved.

The publication of the BUPA schedule during June 1990 stimulated a very strong response. Representations were made to BUPA that the anaesthetic fees were grossly unsatisfactory, especially at the minor end of their range where they were derisory, and in the complex major categories. A letter was sent to all Linkmen and advice given that all members should follow the Association's guidelines. As a consequence of discussion between BUPA executives and representatives of the Association, with effect from October 1990, BUPA is to increase the benefit allocation for anaesthesia for minor surgical procedures in line with the Association's recommended scale of fees. The Association welcomes this development but continues to make vigorous efforts to achieve an early return to the previously accepted levels of benefit for other categories of surgical procedure.

Other problems discussed by the Committee include post-operative analgesia, intensive care, obstetric anaesthesia and chronic pain relief.

21. Junior Anaesthetists' Group

The JAG Committee met five times throughout the year including the Annual General Meeting at Jordanhill College, Glasgow.

Three members of the Committee retired this year, Dr D.L. Paul, Chairman, Dr A.L. Skidmore, Vice Chairman and Junior Linkman Co-ordinator, and Dr C.R.D. Laird, representative on the Anaesthetics Sub-Committee (CCSC) and Assistant Junior Linkman Co-ordinator. We are extremely grateful for all the hard work they have contributed over the years; they will be sadly missed. On behalf of JAG we welcome to the Committee Dr Susan Anderson, Dr Susan Geddes and Dr I. McMenemin.

The Annual Scientific Meeting was held at Jordanhill College, Glasgow, 4-6 April 1990. Both the scientific and social programmes were of exceptional quality and a credit to Dr D.A. Dutton and his Organising Committee. The meeting was sup-

ported by 333 delegates, the largest number ever to attend a JAG ASM, and 20 technical exhibition stands, the largest for over 10 years. The President's Medal was presented to Dr S. Ridley, (Glasgow), and there were two runners up in the Registrars' Prize Competition, Dr Helen Jones, (London), and Dr I. Lewis, (Winchester).

Last year's Junior Linkman Seminar was considered a success and will be repeated this October. The Junior Linkman Scheme continues to grow; we now have almost 100 Junior Linkmen.

A new JAG Careers Stand was completed this year and a revised edition of the 'Careers Guide' is currently in preparation. The Association booklet, 'Handbook for the Trainee Anaesthetist', a long awaited document, will be published shortly. Clarification of the changes made to the first part of the FCAnaes. Examination has accounted for a delay in publishing this document.

This year JAG has initiated a textbook collection scheme intended to retrieve books, which are not dangerously out of date, for distribution to underdeveloped countries. We hope to send the first consignment to Poland this autumn.

Perhaps one of the most significant developments for trainees this year has been the College of Anaesthetists move to seek a separate charter. JAG, representing the future of anaesthesia in this country, was pleased to lend its support to this initiative.

JAG Committee Members have represented trainees on committees and working parties of the Association, the College of Anaesthetists and a number of related bodies. Effective representation of trainees requires regular feedback and a greater input would be much appreciated.

22. Anaesthetics Sub-committee, CCSC

Following the introduction of NHS Medical Indemnity, the Subcommittee (Chairman Dr J. Edmonds-Seal) was concerned as to the way in which anaesthetists may find themselves among the top ten of specialists against whom legal action might be taken. In response to this concern, the Sub-committee approved a document entitled 'Claims Against Anaesthetists' which highlights the major problems concerning anaesthetic practice. This has major implications for training and therefore has been forwarded to the College of Anaesthetists as well as to the Council of the Association. It was pointed out that health authorities were responsible for providing adequate safe facilities and, in line with the guidelines produced by the Association of Anaesthetists, where these were not present anaesthetists should inform management and surgical colleagues that anaesthetic services will be curtailed to a defined safe level. The large number of anaesthetics carried out in general dental practice remains a matter of concern and the setting up of a Department of Health investigation into the facilities and monitoring of such cases was welcomed.

The Department of Health confirmed that study leave is available for clinical assistants at the discretion of health authorities. This information has been published in the *British Medical Journal* and in *Anaesthesia News*. Further information concerning study leave for clinical assistants will be published through the College Tutors.

Concerning the document 'Achieving a Balance', a paper was agreed indicating some matters that require to be taken into consideration when defining the 'safety net level' for departments of anaesthesia. This has been forwarded to the Association of Anaesthetists and the College of Anaesthetists for information.

The Sub-committee has been concerned about the level of dental anaesthetic fees; even the recent increases do not reflect the expertise and value of the anaesthetic services provided. There has been a long and difficult correspondence with the British Dental Association concerning the proportion of fee held back by dental practitioners for the provision of anaesthetic facilities. The matter is not yet resolved.

The BMA produced a document, 'Private Consultant Work: BMA Guidelines 1989', in which the principle of separation of anaesthetists' and surgeons' fees was approved by the Sub-committee. However, since the document contained so many anomalies and did not reflect the current market cost of anaesthetic services at the time of publication, the sub-committee was unable to support the document as published. The document is at present being revised following further advice from the Association's Private Practice Committee, via the CCSC Anaesthetics Sub-committee.

The Sub-committee has made an early appraisal of the new consultant contract and the report of the CCSC Negotiating Sub-committee on the draft circular on this matter. A number of important points were identified and relayed to the Joint Association/College Working Party on consultant workload.

23. Museum, Library and Archives Committee

This Committee (Chairman Dr T.B. Boulton, Honorary Archivist) was formally upgraded from a Sub-committee earlier this year. It now benefits from the co-opted membership of Dr Ghislaine Lawrence (Assistant Curator at the Science Museum) and Dr Aileen Adams (Honorary Archivist, College of Anaesthetists).

An impressive exhibition, 'Painless Tooth Pulling', is currently on display in the Museum. It was mounted with the co-operation of the Wellcome Historical Medical Library, the British Dental Association and other bodies. The problem of museum storage is being addressed in conjunction with the Science Museum. The Association's BOC Museum (Honorary Curator, Dr D.G.A. Wilkinson) now no longer collects large floor-standing machines but liaises with the Science Museum to ensure the preservation of examples brought to the notice of the Curator. The Museum at Bedford Square is seen as having a special function in providing exhibitions and information services for specialist historians of anaesthesia in parallel with the Science Museum's role in relation to the general public.

A policy of careful expansion of the collection of works concerning the historical aspect of anaesthesia is being maintained and a notable addition to the collection this year was a group of over 100 books selected by Dr I. McLellan (Honorary Librarian) from the personal library of Dr J. Alfred Lee and donated by his widow and son. There was no active demand for reference to the wide range of current journals which had been maintained at 9 Bedford Square. Provision will be limited in future to *Anaesthesia*, the *British Journal of Anaesthesia*, the *British Medical Journal* and the *Lancet*. The film collection continues to develop as a unique and valuable historical resource, although recent changes in the law of copyright impose some limitations on the use of the material.

Dr Audrey Eccles (ICI Archivist/Librarian) continues her invaluable work in co-ordinating and developing the various functions with which the Committee is entrusted.

24. Specialist Societies of Anaesthesia

The Annual Meeting between Officers and Representatives of Specialist Societies was held on 17 November 1989. The meeting took its usual format; the President reviewed the Association's current activities and each representative gave a brief resumé of their Society's organisation and areas of activity.

The representatives of the Specialist Societies agreed that in future each society, on a rotational basis, would organise a session at the Association Annual Winter Scientific Meeting. The Association of Paediatric Anaesthetists agreed to organise the first of these sessions at the 1991 Winter Meeting.

Concern had been expressed at the increase in pressure being placed on both trainee and senior anaesthetic staff in their attempts to obtain expenses from health authorities to enable them to attend national meetings. The meeting discussed a proposal that major Association and Specialist Society meetings should be run back to back or, alternatively, that major meetings be extended to include specialist meetings. Representatives were invited to refer this matter for discussion with their respective committees and submit their views formally to the Association. The Association has also recently sought the views of the Specialist Societies on the concept of the Association setting up a register of future meetings, the intention being that any body planning a meeting would register at the earliest opportunity to enable other to check before they finalise their dates. The Association already keeps a five-year diary for this purpose.

25. Working Parties

Workload for Consultant Anaesthetists (Chairman Dr M.M. Burrows, President). The Joint Working Party was set up in June 1989 to consider the workload of consultant anaesthetists and

update the Association's recommendations last published in 1983. During the first few months of 1990 the activities of the Working Party were suspended pending the outcome of negotiations on the consultant contract being held at national level. Thereafter, some amendments were made prior to submission of a draft document to the Council of the Association and to Council of the College. On obtaining the approval of both Councils, the document, 'Workload for Consultant Anaesthetists', was published in mid-July and distributed to consultant members of the Association, Linkmen, REAs, College Tutors and health authority managers.

The document is intended to clarify for consultant anaesthetists their contractual commitments to the NHS and also to serve as a guide for health authorities who are either holding the contract or administering it.

Assistance for Anaesthetists (Chairman Dr M.M. Burrows, President). The Working Party has welcomed the responses to its work by national bodies. The improved pay and regrading structure for ODAs within the medical technician scales has been implemented. The flexibility built into this structure has allowed management to reward ODAs more realistically, and feedback to the working parties suggests that seniors are being placed on the MT2 and MT3 scales. Credible pay allows ODAs to consider the very real long-term advantages of NHS employment and there are signs that recruitment and retention are improving. The Bevan Report, 'Management and Utilisation of Operating Departments', has recommended registration of ODAs and an examination of possibilities for combined training of non-medical operating department staff. The Report's recognition of the need for skilled assistance as a part of the anaesthetic team is particularly welcome. The Working Party is seeking to encourage implementation of these recommendations and is to meet with representatives of the English National Board regarding training of nurses in anaesthetic related areas.

The Working Party continues to be represented on the Steering Group overseeing the implementation of a National Vocational Qualification in Operating Department Practice. A pilot project, which will replace the current City & Guilds Certificate 752, is in progress at both basic and extended levels and it is possible that the new, competence based, training, which lays great emphasis on workplace assessment, will be introduced early in 1991.

Thanks are due to all members who have communicated their local experience, particularly in the field of training and grading.

Pre-Anaesthetic Check Procedures (Chairman Professor A.P. Adams). The Working Party has received various comments following distribution of the draft document presented to the Linkman Conference in September 1989. These, together with reports from medico-legal sources and from Coroner's inquests, have been considered. The Working Party's draft document, 'Checklist for Anaesthetic Machines', was approved by Council and is now published.

High Dependency Units (Chairman Dr J.A.W. Wildsmith). The Working Party, a multidisciplinary group, has met on three occasions and a draft report was prepared. The aim of the report is to indicate why and how a high dependency facility will improve standards of care in hospital. The report will also outline how such a unit may be established and run.

Radio-Allergosorbent Testing (Chairman Dr W.S. Nimmo). Following a fatal accident inquiry in Aberdeen, the Association was asked to give advice about whether routine screening for specific drug antibodies should be carried out prior to anaesthesia. The Working Party has reviewed the information available about anaphylactic reactions associated with anaesthesia and has considered the advice to be given about such reactions.

The Working Party gathered opinion from experts throughout the world and produced an interim report in September 1989. The full report, including definitions of the terms used, investigation of anaphylaxis and an appendix addressing the suggested management of acute major anaphylaxis under general anaesthesia, will be available at the Annual General Meeting.

Services for Chronic Pain Relief (Chairman Dr J.E. Charlton). The Joint Working Party of the Association of Anaesthetists, College of Anaesthetists and the Intractable Pain Society has continued to meet throughout the year. An interim report to the College of Anaesthetists suggested an increase in the teaching of pain relief in both Basic Training and Higher Specialist Training.

The Working Party has completed a major survey of the working practices in pain relief clinics in the South-Western, North-East Thames, Yorkshire and Northern Regions. The Working Party is

grateful to those members who took part and, in particular, to Dr A.W. Diamond who undertook the difficult task of collating and analysing the complex data.

The Working Party report will be sent for consideration by the various bodies concerned later in 1990.

Acute Adverse Drug Reaction Reporting (Chairman Professor C.J. Hull). This is a joint Working Party of the Association of Anaesthetists and the College of Anaesthetists.

The Committee on Safety of Medicines has agreed to continue the study and is satisfied with its progress. In the period September 1988 to December 1989, 125 anaesthetic forms were returned, inferring that 66% of incidents were still being reported on the standard yellow card. In future when this happens the CSM will send out an anaesthetic yellow form requesting a further report. The form will be amended next year to facilitate computer analysis. A draft report is in progress. A poster advertising the scheme, together with a supply of forms, will be distributed to anaesthetic departments. This will enhance the current arrangement whereby forms are available from the hospital pharmacy. Further information will be disseminated in the College Newsletter, *Anaesthesia News* and by College Tutors.

26. Anaesthesia Management News

This bi-annual Association publication, edited by Professor M.D. Vickers, Cardiff, continues to highlight anaesthetic matters with implications for management. The publication is circulated to Linkmen and District General Managers, but Council recently agreed that the circulation should be extended. Professor Vickers would welcome feedback on this publication.

27. Resuscitation Council

The Council (Association Representative Dr P.J.F. Baskett) has published guidelines for Basic and Advanced Life Support in the British Medical Journal, August 1988, and revised wall posters, flip charts and the 'Resuscitation for the Citizen' booklet.

During the coming year it is planned to introduce Advanced Cardiac Life Support (ACLS) courses and at present the course and instruction manual are being written. A Paediatric Sub-committee has been formed and plans are in hand to produce guidelines for the resuscitation of children. It is also planned to formulate guidelines for the introduction and use of semi-automatic defibrillators.

28. The Monospecialist Committee for Anaesthesia and Re-animation

Two meetings of the Monospecialist Committee (Association Representative Dr W.R. MacRae, Honorary Treasurer) were held during the year. Continuing attention was given to matters of duration of training and assistance for anaesthetists.

It became clear that although the committee had agreed several years ago to recommend to the main UEMS committee a minimum training period in anaesthesia and related subjects of five years before recognition as a specialist anaesthetist, this information had not been widely disseminated through the countries of the European Community and appropriate legislative action had not been enacted by the European Parliament. The committee therefore decided to raise the matter again urgently with the main UEMS Committee.

Detailed matters of nomenclature of posts were also debated in an attempt to further the chances of a smooth period of harmonisation before 1992.

Composition of Council, its Sub-Committees and Working Parties

Council

Five meetings held (attendance in brackets).

Officers: Dr M.M. Burrows, President (5); Dr P.J.F. Baskett, President Elect (5); Professor M. Rosen, CBE Immediate Past President (4); Dr J.F. Nunn, Vice-President (5); Dr P. Morris, Vice-President (4); Air Cmdr C.A.B. McLaren, Vice-President (3);

Dr W.R. MacRae, Honorary Treasurer (5); Dr W.L.M. Baird, Honorary Secretary (5); Dr J.E. Charlton, Assistant Honorary Secretary (5); Dr J.N. Lunn, Editor (5).

Elected Members: Dr Lauren G. Allan (5); Dr D.S. Arthur (3); Dr J. Edmonds-Seal (5); Dr Margaret L. Heath (5); Professor P. Hutton (5); Dr S.M. Lyons (5); Dr I. McLellan (4); Dr R.L. Marks (5); Dr R.S. Vaughan (5); Dr R.M. Weller (5); Dr J.A.W. Wildsmith (3); Dr D.L. Paul, JAG (4); Dr P.J. Heath, JAG (5).

Co-opted Members: Surg Commander S.B. Merrill, Armed Forces Representative; Dr D.C. Moriarty, Dean, Faculty of Anaesthetists, RCS in Ireland; Dr D. Riordan, Convenor, Irish Standing Committee; Professor M. Rosen, President, College of Anaesthetists; Professor M.K. Sykes, Advisor on Anaesthetics to CMO; Professor M.D. Vickers, President, European Academy of Anaesthesiology; Dr J.S.M. Zorab, President, WFSA.

Advisory Committee

Five meetings held (attendance in brackets).

Chairman: Dr M.M. Burrows (5); Dr W.L.M. Baird (5); Dr P.J.F. Baskett (4); Dr J.E. Charlton (5); Dr Margaret L. Heath (3); Professor P. Hutton (4); Dr J.N. Lunn (5); Dr W.R. MacRae (4); Professor M. Rosen (4); Dr J.A.W. Wildsmith (2); Dr D.L. Paul, JAG (5).

Editorial Board

Chairman: The President; *Editor:* Dr J.N. Lunn; *Associate Editor:* Dr M. Morgan; *Assistant Editors:* Professor A.R. Aitkenhead, Dr R. Greenbaum, Dr D.E.F. Newton, Dr Rosemary Mason (from June 1990), Dr C.F. Scurr.

Representatives of Council: Dr P.J.F. Baskett, Dr J. Edmonds-Seal, Air Commodore C.A.B. McLaren, Dr H. St John Grey, JAG.

Co-opted Members: Mr E. Warburton, Honorary Financial Adviser, Mrs Joan Fujimoto, Academic Press, Miss Jane Lawrence, Academic Press.

Education and Research Committee

Dr J.A.W. Wildsmith (*Chairman*), Professor P. Hutton, Dr S.M. Lyons, Dr I. McLellan, Dr J.F. Nunn, Dr R.S. Vaughan, Dr D.L. Paul, JAG (until April 1990), Dr Sue Anderson, JAG (from 1990).

Safety Committee

Professor P. Hutton (*Chairman*), Professor A.P. Adams, Chairman, WFSA Safety Committee, Dr Lauren G. Allen, Dr D.S. Arthur, Dr R. Greenbaum, Dr Margaret L. Heath, Air Cdre C.A.B. McLaren, Dr R.L. Marks, Dr P. Morris, Dr N.I. Newton, Professor M.K. Sykes, Dr P.W. Thompson, Dr R.M. Weller, Dr P.J. Heath, JAG, Dr Kate Allsop, Medical Defence Union, Mr L.W.M. Arrowsmith, Department of Health, Mr C. Bray, Department of Health, Dr Juliet M. Davies, Medical Protection Society, Mr M. Freeman, Chairman, BAREMA Dr I.G. Simpson, Joint Co-ordinating Committee of Medical Defence Organisations, Dr Helen Sutton, Department of Health.

International Relations Committee

Dr S.M. Lyons, Chairman, Dr M. Dobson, Dr R.J. Eltringham, Dr M.T. Inman, Dr R.L. Marks, Dr P. Morris, Dr D.A. Saunders, Bernard Johnson Adviser, Dr R.M. Weller, Dr J.A.W. Wildsmith, Dr J.S.M. Zorab, Dr M.W. Platt, JAG.

Representatives from:

Overseas Development Administration, Bureau for Overseas Medical Service, British Council, Inter-varsity Council, Committee for International Co-operation in Higher Education, Voluntary Services Overseas, Appropriate Health Resources and Technologies Action Group.

Finance Committee

The President (*Chairman*), Dr J. Edmonds-Seal, Dr I. McLellan, Dr R.L. Marks, Professor M. Rosen, Mr E. Warburton, (*Honorary Financial Adviser*).

Private Practice

Dr R.M. Weller (*Chairman*), Dr Lauren G. Allan, Dr D.S. Arthur, Dr P.J.F. Baskett, Dr J. Edmonds-Seal, Dr E.B. Lewis, Dr R.S. Vaughan, Dr I. Verner.

Working Party on Assistance for Anaesthetists

Dr M.M. Burrows (*Chairman*), Dr Margaret L. Heath (*Secretary*), Dr D.S. Arthur, Dr J. Edmonds-Seal, Dr S.M. Lyons, Dr P. Morris, Professor M.D. Vickers.

Working Party on High Dependency Units

Dr J.A.W. Wildsmith (*Chairman*), Dr S.M. Lyons, Dr M.G. Pearson, Consultant Physician, (*Liverpool*); Dr G.A.D. Rees, Consultant Anaesthetist, (*Cardiff*); Dr Gillian Hanson, Intensive Care Society; Dr G.S. Routh, Intensive Care Society; Mr G.G. Youngson, Consultant Surgeon, (*Aberdeen*); Mrs R. Wilkinson, RCN; Dr D.L. Paul, JAG.

Specialist Societies

President, (*Chairman*).

Representatives from:

Age Anaesthesia Association, Association of Cardiothoracic Anaesthetists, Association of Dental Anaesthetists, Association of Paediatric Anaesthetists of Great Britain and Ireland, College of Anaesthetists, Computing in Anaesthesia Society, European Society of Regional Anaesthesia—UK Committee, History of Anaesthesia Society, Intractable Pain Society of Great Britain and Ireland, Junior Anaesthetists' Group, Neuroanaesthetists' Travelling Club, Obstetric Anaesthetists' Association, Plastic Surgery and Burns Anaesthesia.

Working Party on Pre-anaesthetic Check Procedures

Professor A.P. Adams (*Chairman*), Dr P. Bickford-Smith, Dr J.D. Henville, Dr N.I. Newton, Dr P. Morris, Dr M.W. Platt, JAG.

Joint Working Party on Services for Chronic Pain Relief

Dr J.E. Charlton (*Chairman*), Dr A.W. Diamond, Intractable Pain Society (*Secretary*), Dr R.L. Marks, Association of Anaesthetists; Dr K. Budd, Intractable Pain Society; Dr F.R. Ellis, College of Anaesthetists; Dr P.J.D. Evans, Intractable Pain Society; Dr D.J. Hatch, College of Anaesthetists.

Working Party on Pre-anaesthetic Screening Tests for Antibodies to Anaesthetic Agents

Dr W.S. Nimmo (*Chairman*), Professor A.R. Aitkenhead, Dr R.M. Weller, Professor R.S.J. Clarke.

Joint Association/College Working Party on Workload for Consultant Anaesthetists

President, Association of Anaesthetists (*Chairman*), Dr R.S. Atkinson, College of Anaesthetists; Dr P.J.F. Baskett, College of Anaesthetists; Dr P. Morris, College of Anaesthetists; Professor J. Norman, College of Anaesthetists; Dr J. Edmonds-Seal, CCSC; Dr E.B. Lewis, CCSC.

Anaesthesia News Management Board

Dr M.M. Burrows (*Chairman*), Dr Margaret L. Heath, Dr J.N. Horton (*Editor*).

Working Party on the Role of the Anaesthetist in the Emergency Service

Dr P.J.F. Baskett, President Elect, (*Chairman*), Dr C.R.D. Laird, JAG; Professor J. Norman, College of Anaesthetists; Professor P. Hutton, Dr R.M. Weller, Dr Ann Sutcliffe, Dr R. Sleet, Mr D. Skinner.

The Executive Officers of the Association are ex-officio Members of all Committees and working Parties above

Manpower Advisory Panel

Dr J. Edmonds-Seal, Dr Elizabeth Spencer, JAG.

City and Guilds (ODA Training Committee)

Dr Margaret L. Heath.

Joint Committee for Higher Training of Anaesthetists

Dr W.L.M. Baird, Dr J.A.W. Wildsmith, Dr P.J. Heath, JAG.

Trustees of the Anaesthetists Academic Foundation

President, Association of Anaesthetists.

National Confidential Enquiry into Perioperative Deaths

Dr M.M. Burrows, Association of Anaesthetists; Dr J.N. Lunn, Association of Anaesthetists.

Joint Association/College Working Party on Adverse Drug Reactions Reporting

Association representatives: Professor A.R. Aitkenhead, Dr W.L.M. Baird, Dr J.A.W. Wildsmith. *College representatives:* Professor A.A. Spence, Professor C.J. Hull, Professor J. Norman.

Museum, Library and Archives Sub-committee

Dr T.B. Boulton, Honorary Archivist and Chairman, Dr D.J. Wilkinson, Honorary Curator, Charles King Collection of Historical Apparatus; Dr I. McLellan, Honorary Librarian; Dr Audrey Eccles, ICI Archivist/Librarian; Dr Ghislaine Lawrence, Wellcome Museum.

Anaesthetics Sub-committee, CCSC

Dr J. Edmonds-Seal (*Chairman*), Dr Lauren G. Allan, Dr W.L.M. Baird, Dr S.M. Lyons, Dr S. Geddes, JAG.

Junior Anaesthetists' Group

Dr D.L. Paul (*Chairman*) (until April 1990); Dr P.J. Heath (*Chairman*) (from April 1990); Dr Elizabeth Spencer (*Secretary*) (from April 1990); Dr A.L. Skidmore (*Vice Chairman*) (until April 1990); Dr P. Morris (*Council representative*); Dr C.R.D. Laird (until April 1990); Dr I. McMenamin; Dr J. Owens; Dr M.W. Platt; Dr H. St John Gray; Dr J. Shephard; Dr Susan Anderson (from April 1990); Dr Susan Geddes (from April 1990).

Technical Exhibitions Co-ordinator

Dr R.S. Vaughan.

Association Exhibition Stand Co-ordinators

Dr Wendy E. Scott, Dr R. Jane Chestnut.

UEMS Representative

Dr W.R. MacRae.

Resuscitation Council, UK

Dr P.J.F. Baskett.

Postgraduate Study Day

Dr J.A.W. Wildsmith, Association of Anaesthetists; Dr J.F. Nunn, Association of Anaesthetists; Professor C.J. Hull, College of Anaesthetists; Dr F.R. Ellis, College of Anaesthetists.

Association Representative on College of Anaesthetists Audit Committee

Dr J.E. Charlton.

Association Representatives on College of Anaesthetists Manpower Advisory Panel

Dr Margaret L. Heath, Dr J. Edmonds-Seal, Professor M.K. Sykes. (Working Group to review unfilled consultant posts).

Association Representative on College of Anaesthetists Fund Raising Committee

Dr W.L.M. Baird.

Association Representative on National Health Service Training Authority ODA Training Project (NHSTA)

Dr Margaret L. Heath.

Anaesthesia Management News

Professor M.D. Vickers (*Editor*), Dr Lauren G. Allan.

Future Meetings

1990—*Postgraduate Study Day, 13 October, Royal College of Surgeons.

—American Society of Anaesthesiologists (ASA) Annual Meeting 19–23 October.

1991—*Winter Scientific Meeting and Technical Exhibition 18–19 January Queen Elizabeth II Conference Centre, London.

—*Junior Anaesthetists' Group Annual Scientific Meeting and Linkman Conference, 3–5 April John Radcliffe Infirmary, Oxford.

—7th World Congress on Emergency and Disaster Medicine 13–16 May Montreal, Quebec, Canada.

—History of Anaesthesia Society, 31 May–1 June Rotterdam.

—3rd European Congress Paediatric Anaesthetists, 31 August–3 September Liverpool.

—*Annual Scientific Meeting, Linkman Conference and Technical Exhibition, 11–13 September Harrogate Conference Centre (to be organised by Leeds).

- 1992—*Winter Scientific Meeting and Technical Exhibition 10-11 January Queen Elizabeth II Conference Centre, London.
 —3rd International Symposium on the History of Anaesthesia, 29 March-2 April Atlanta, Georgia.
 —*Junior Anaesthetists' Group Annual Scientific Meeting and Linkman Conference, 1-3 April The Victoria Rooms, Bristol.
 —European Society of Regional Anaesthesia Meeting 10-13 June Brussels.
 —10th World Congress of Anaesthesiology, 12-19 June The Hague.
 —*Annual Scientific Meeting, Linkman Conference and Technical Exhibition, 9-11 September Bournemouth International Centre (to be organised by Liverpool).
 —History of Anaesthesia Society, 7 November Cambridge.
- 1993—*Junior Anaesthetists' Group Annual Scientific Meeting and Linkman Conference, April Sheffield.
 —History of Anaesthesia Society, 23-24 June North Wales.
 —*Annual Scientific Meeting, Linkman Conference and Technical Exhibition, Scottish Exhibition and Conference Centre, 22-24 September Glasgow. Joint meeting with Canadian Anaesthetists' Society (CAS).
- 1994—Junior Anaesthetists' Group Annual Scientific Meeting and Linkman Conference, April Newcastle upon Tyne.
 —*Annual Scientific Meeting, Linkman Conference and Technical Exhibition, 7-9 September Brighton Conference Centre.
 —9th European Congress, European Section, WFSA, 2-7 October Jerusalem.
- 1995—*Junior Anaesthetists' Group Annual Scientific Meeting and Linkman Conference, April Leicester.

*Association meeting.

Association Linkmen

Names are grouped according to Regional Health Authority (Area in Scotland); the order of names within each group is arbitrary and without significance.

England

Northern (R.H.A. 1)

D.J.H. Daniel (*Hartlepool*); B. Dennison (*South Tees*); R. Goodwin (*Durham*); D.C. Townsend (*South West Durham*); K.S. Cameron (*North West Durham*); C. Beeton (*Darlington*); I. Anderson (*Northumberland*); M.R. Bryson (*Newcastle Central Sector*); S.K. Greenwell (*North Tyneside*); R.P. Harpin (*Newcastle Western Sector*); P. Copeland (*Newcastle Eastern Sector*); G. Harris (*North Tees*); D.F. Jones (*Cumberland Infirmary, East Cumbria*); R.M. Freeman (*Gateshead*); G. Earnshaw (*Barrow-in-Furness*); R. Gautam (*South Tyneside*); M.K. Johnson (*Sunderland*); J.N. Hodgkinson (*South Cumbria*); D.M. Watson (*West Cumbria*).

Yorkshire (R.H.A. 2)

T.D. Biswas (*Pontefract*); S. Firn (*Wakefield*); S. Swindells (*St James' University Teaching Hospital, Leeds East*); J.S. Gibson (*Leeds General Infirmary*); M. Bembridge (*Huddersfield*); S. Siddiqui (*Halifax*); J.A. Robertson (*Airedale*); G.J. MacWhirter (*Harrogate*); R.G. Wheatley (*York*); G.T. Whitfield (*Scarborough*); T.I.R. Williams (*Hull*); J.M. Clark (*Grimsby*); P.J. Bickford-Smith (*Bradford*); M. Evans (*Dewsbury*).

Trent (R.H.A. 3)

D.P. Cartwright (*Derby Hospitals*); J.V.B. Mundy (*Royal Hallamshire Hospital, Sheffield*); J. Hunsley (*Northern General Hospital, Sheffield*); P.J. Mawson (*Rotherham*); B.R. Milne (*Don-*

caster); P.S. Gadgil (*Barnsley*); C.J. Bowley (*Nottingham*); J.S. Mason (*Mansfield*); J.F.W. Thompson (*Scunthorpe*); A.H.R. Spong (*Lincoln*); A.G. Cole (*Leicester*); M.J. Wolfe (*Chesterfield*); P. Smith (*Worksop*); M. Bexton (*Pilgrim Hospital, Boston, S Lincs*); P.T. Bull (*Central Notts*); V. Boyd (*Rotherham*).

East Anglia (R.H.A. 4)

P.J. Keep (*Norwich*); J.R.O. Jenkins (*Great Yarmouth*); R.N. Francis (*King's Lynn*); P. Benison (*Huntingdon*); C.W. Glazebrook (*Cambridge*); A.P. Gallimore (*West Suffolk*); J.M.F. Hartley (*Peterborough*); D.J. Elliott (*Ipswich*).

North West Thames (R.H.A. 5)

K.P. Kyriakou (*Ealing*); D.J.C. Robinson (*St Mary's*); R.L. Hargrove (*Westminster*); P. Chakrabarti (*West Middlesex*); M. Meurer-Laban (*King Edward VII, Ealing*); J. Lumley (*Hammer-smith*); M.T. McGibbon (*Hillingdon*); P. Forrester (*Charing Cross*); K.M. Konieczko (*Northwick Park*); E.S. Walsh (*Central Middlesex*); A.E. Nichol (*Barnet*); S. Jothilingham (*Stevenage*); M.I. Carter (*Luton*); J. Hurst (*Bedford*); G.M. O'Sullivan (*Wat-ford*); Z.P. Shah (*Welwyn Garden City*); M.J. Boscoe (*Harefield Hospital*); N. Poobalasingam (*N. Middlesex*); W.J. Rickford (*N. Hertfordshire*); B.M. Dempsey (*Ashford, Middlesex*); N. Fletcher (*Edgware*).

North East Thames (R.H.A. 6)

A.R. Deacock (*Royal National Orthopaedic*); J. Ormrod (*Univer-sity College Hospital*); D.J. Wilkinson (*St Bartholomew's Hospital*); J.D. Hill (*West Essex*); P.J. O'Shea (*The London Hospital*); S.A.I. Helwa (*Enfield*); M.S. Pegg (*Royal Free*); P.M. Bashir (*Barking*); G.B. Rushman (*Southend*); P.O. Bodley (*Romford*); R.W. Griffin (*Colchester*); R.A. Spilsbury (*Chelmsford*); J.L. Handy (*Broomfield Hospital, Mid Essex*); D.A. Thomas (*Harold Wood Hospital*); E.P. Gibbs (*Basildon*); E.B. Javed (*Barkingside*); S.K. Hepton (*Old-church, Romford*); D.V.A. Kalmanovitch (*Whittingdon*).

South East Thames (R.H.A. 7)

B.C. Bradburn (*Kent and Canterbury*); A.R. Thompson (*Hastings*); B. Steer (*Eastbourne*); E.A. Proctor (*Canterbury and Thanet*); E.B. Lewis (*Ashford, Kent/S.E. Kent*); K.H.P. Singh (*Dartford*); P.E. Daly (*Greenwich*); M.A. Thompson (*Lewisham, North Southwark & Guy's Hospital*); I.M. Corall (*King's College*); D.A. Young (*Bromley*); J.A. Mathias (*St Thomas', London*); C.J. Barham (*East Grinstead*); D. Simpson (*Medway*); J.M. Lamberty (*Brighton*); D. Iyer (*Maidstone*).

South West Thames (R.H.A. 8)

I.K. Stanley-Jones (*St Peters, Chertsey*); W.A.L. Rawlinson (*Cuck-field*); R.C. Brookes (*Croydon*); J.R. Fozard (*Guildford*); D.A. Wright (*Kingston upon Thames*); H.R. Waters (*Worthing*); J.C. Missen (*Chichester*); W.A. Lindsay (*St Helier Hospital, Merton & Surrey*); A.C. Thurlow (*St George's London*); A.M. Rollin (*Epsom*); I.A. Sutherland (*Frimley Park*); D.A. Young (*Caterham*); R.M.H. Ratcliffe (*East Surrey Hospital*); L. St John Jones (*Craw-ley*); C.J. Moon (*Crawley*).

Wessex (R.H.A. 9)

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The Committee on Enzymes of the Scandinavian Society for Clinical Chemistry and Clinical Physiology. Recommended method for the determination of gamma-glutamyltransferase in blood. *Scandinavian Journal of Clinical Laboratory Investigation* 1976; **36**: 119–25.

Anonymous. Epidemiology for primary health care. *International Journal of Epidemiology* 1976; **5**: 224–5.

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OSLER AG. Complement: mechanisms and functions. New York: Prentice-Hall, 1976.

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American Medical Association Department of Drugs. *AMA drug evaluations*, 3rd edn. New York: Publishing Sciences Group, 1977.

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RHODES AJ, VAN ROOVEN CE, comps. *Textbook of virology: for students and practitioners of medicine and other health sciences*, 5th edn. Baltimore: The Williams & Wilkins Co., 1968.

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National Center for Health Statistics. Acute conditions: incidence and associated disability, United States, July 1968–June 1969. Rockville, MD: National Center for Health Statistics, 1972. (*Vital and health statistics, Series 10: Data from the National Health Survey*. No. 69) [DHEW publication No. (HSM) 72-1036].

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SHAFFER RA. Advances in chemistry are starting to unlock mysteries of the brain: discoveries could help to cure alcoholism and insomnia, explain mental illness. How the messengers work. *Wall Street Journal* 1977 Aug 12: 1(col 1), 10(col 1).

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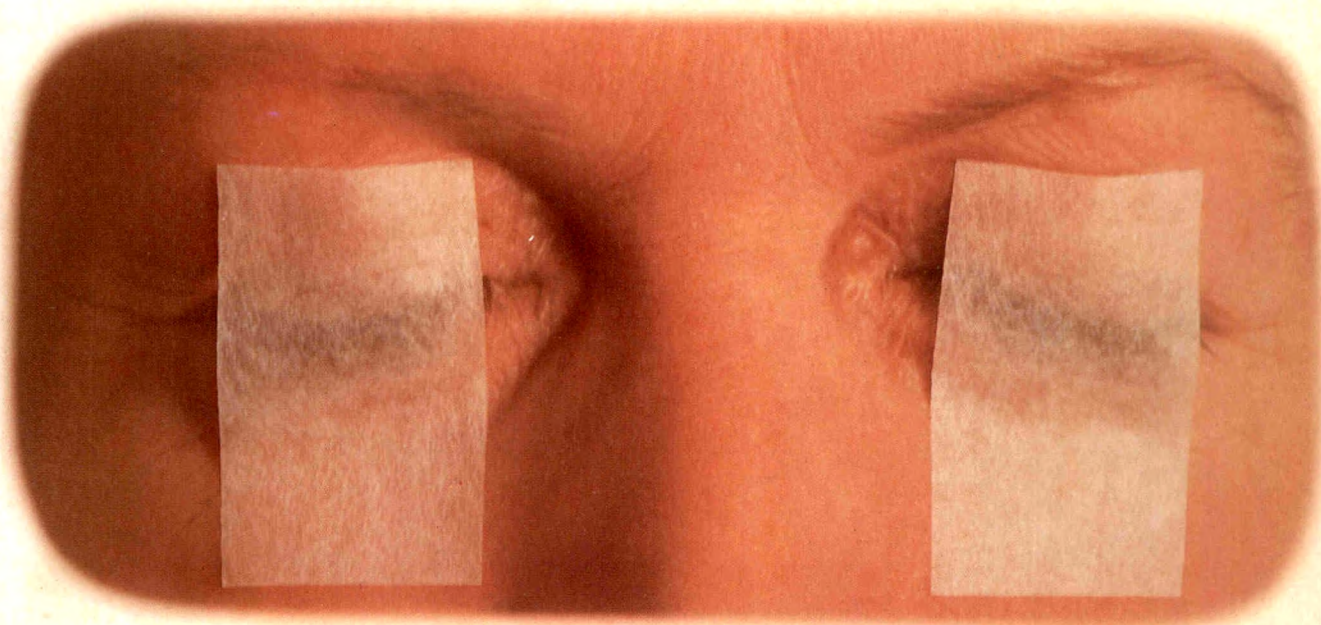
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